THE American JOURNAL OF Surgery

SEPTEMBER 1954

THURSTON SCOTT WELTON, M.D., EDITOR

Surgical Review of Hand Surgery

Pylephlebitis and Liver Abscess

Neoplasms of Small Intestine

SIXTY-THIRD YEAR OF PUBLICATION

SEPTEMBER 1954

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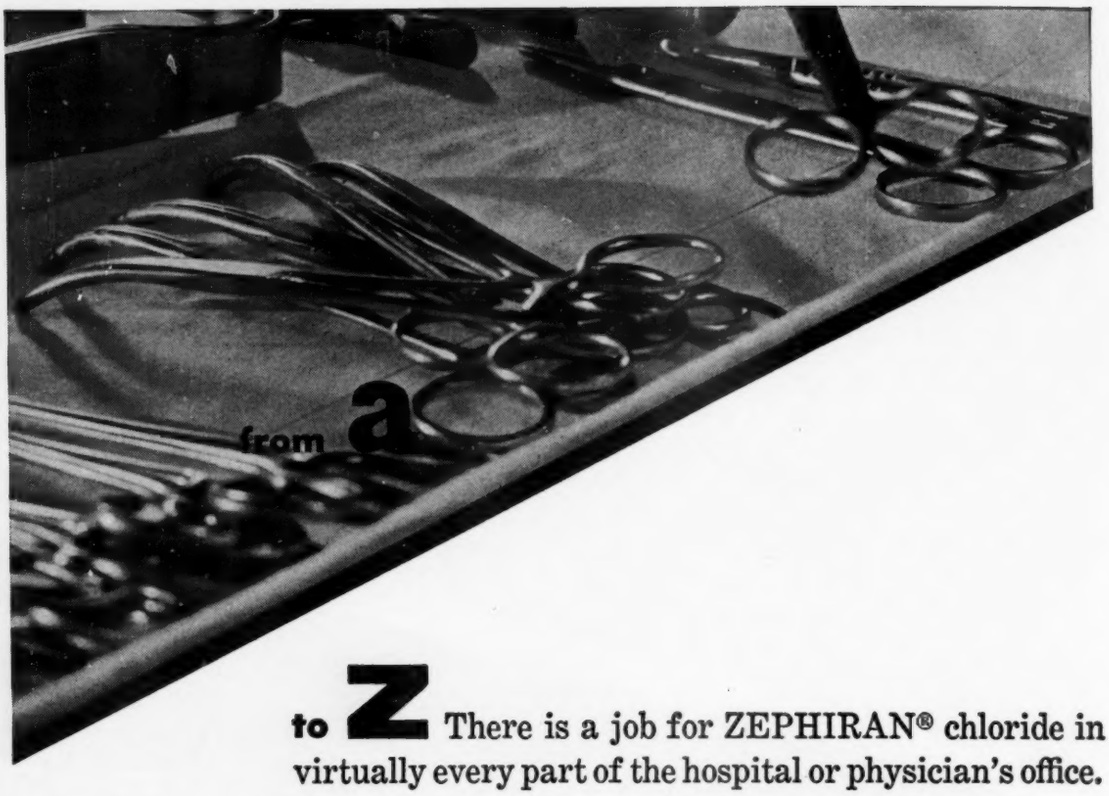

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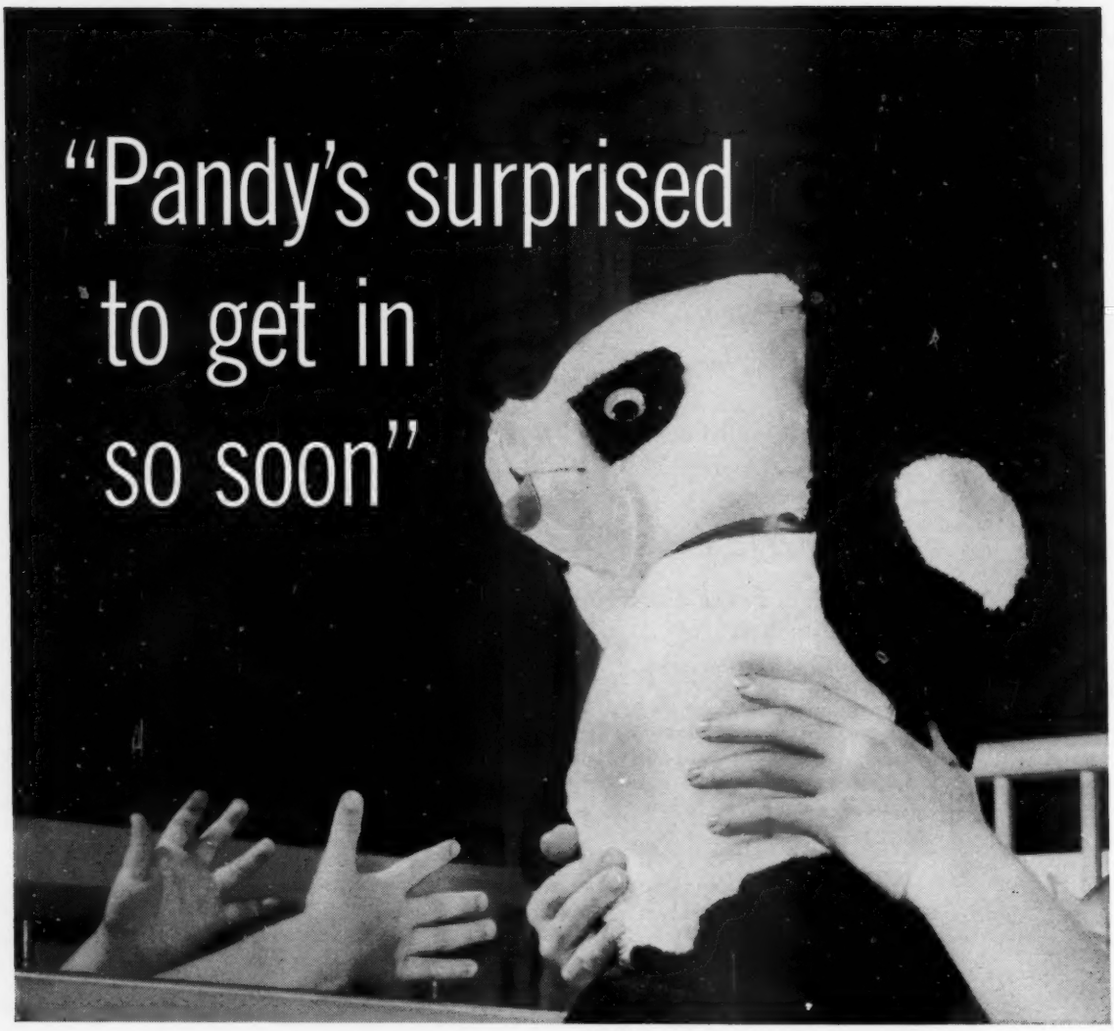

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1. Brown, S., and Fehlman, B. F.: Missouri Med. 50:609 (Aug.) 1953.
2. Stephen, C. R., Nowill, W. K., and Martin, R.: Anesthesiology *13*:646 (Nov.) 1952.
3. Flowers, C. E., Jr.: Am. J. Obst. & Gynec. 65:1027 (May) 1953.

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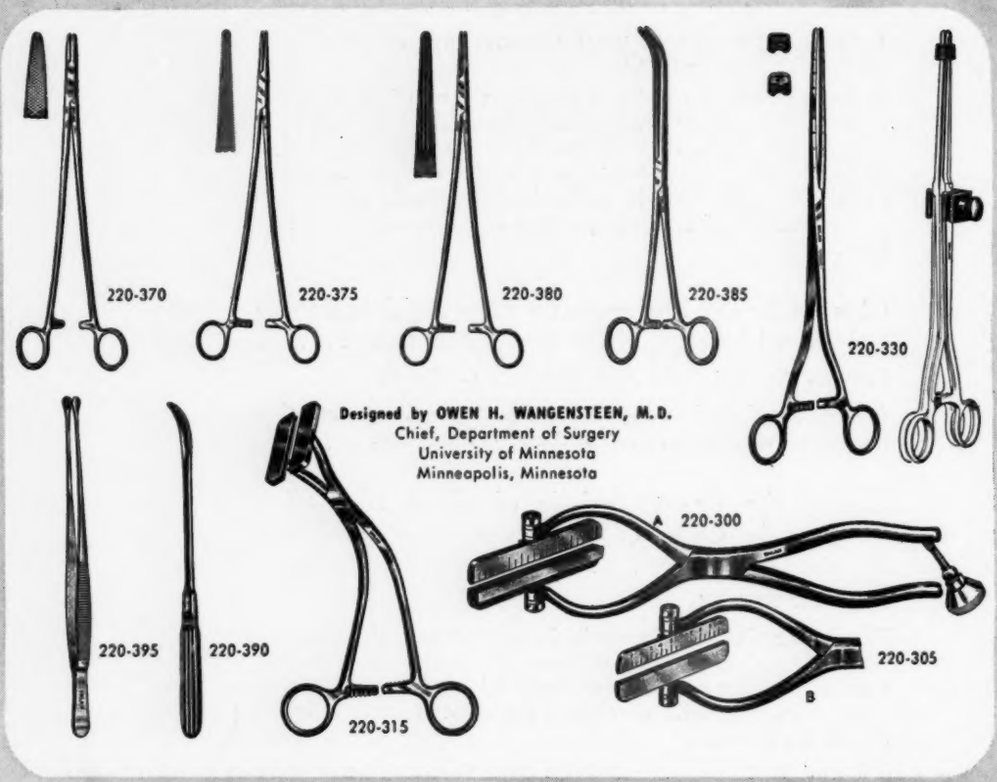

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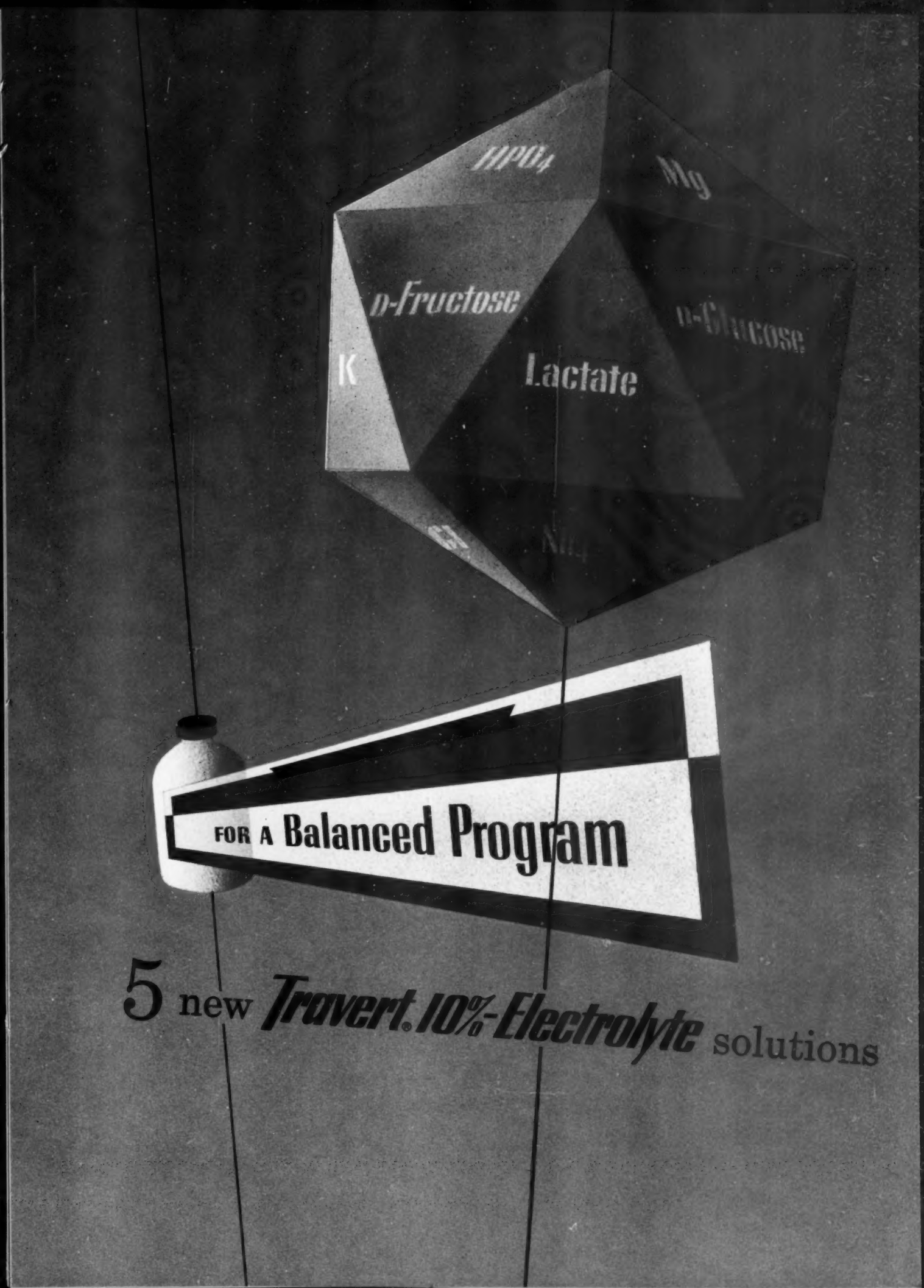

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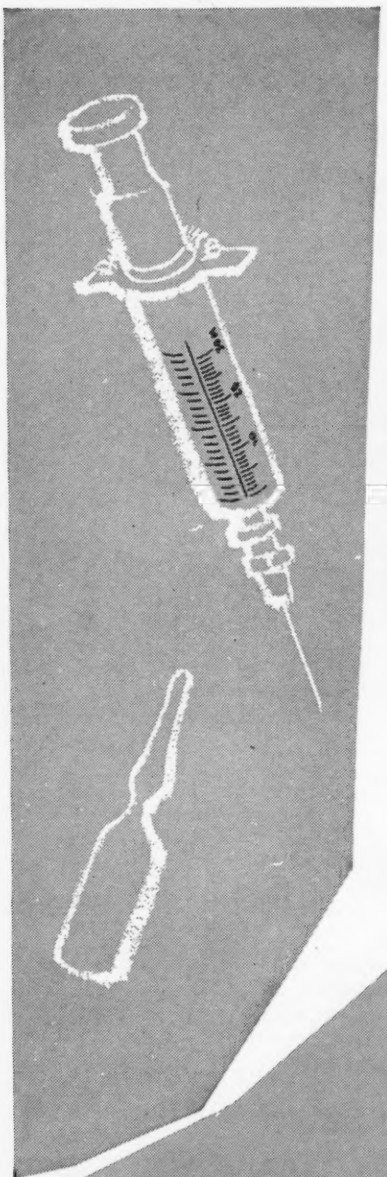

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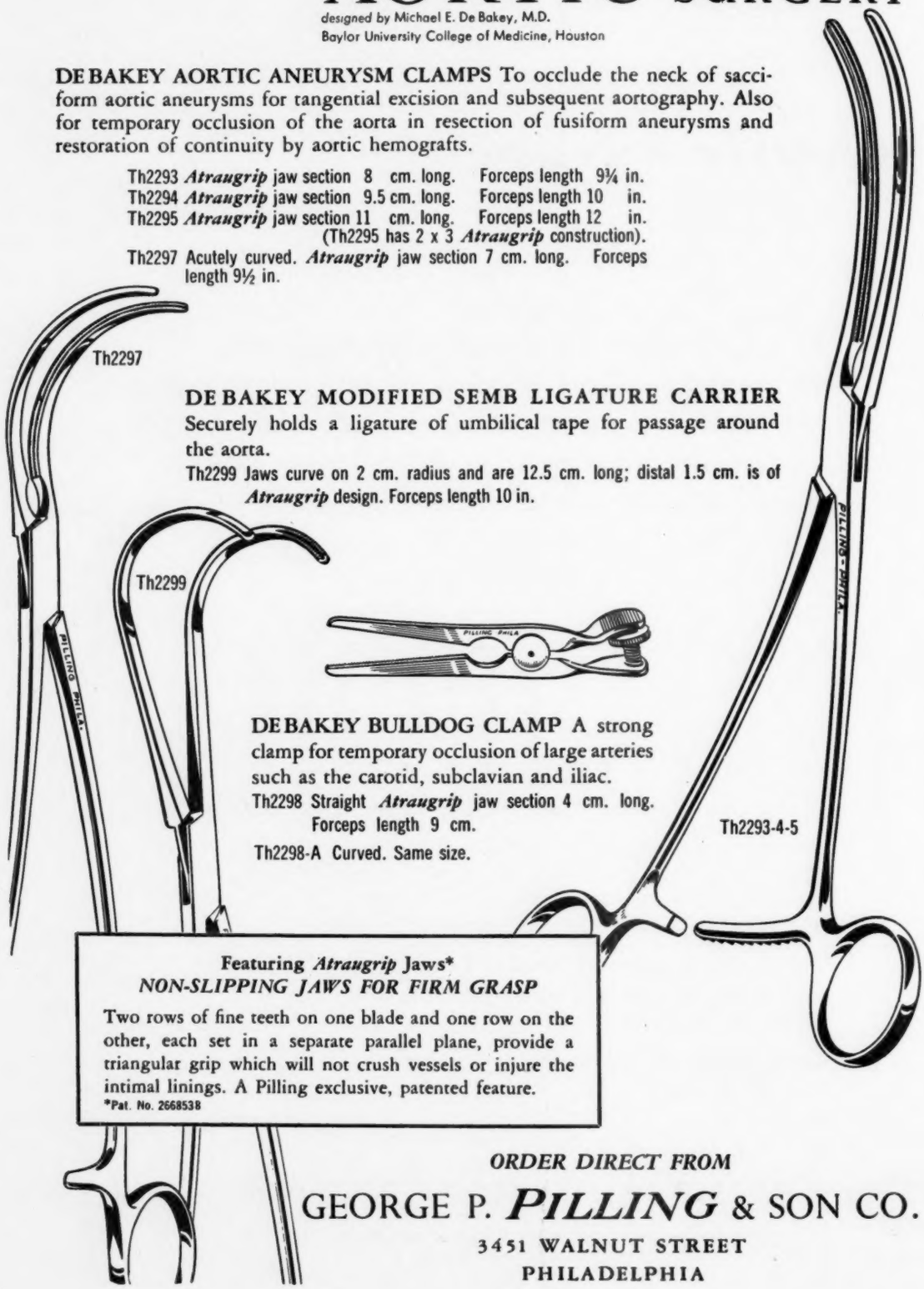

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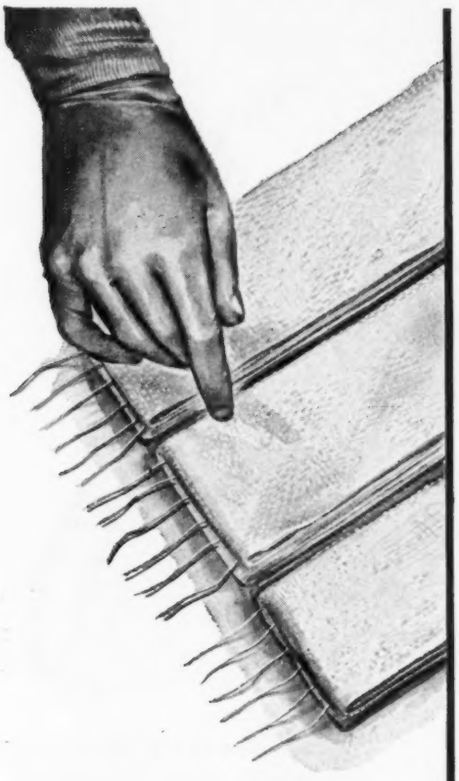

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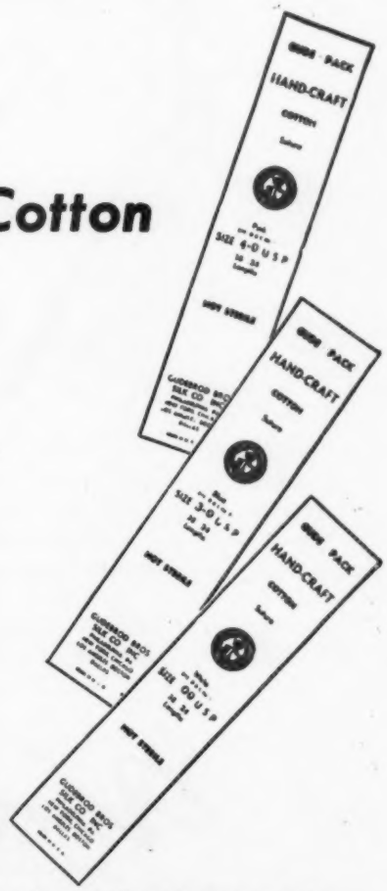

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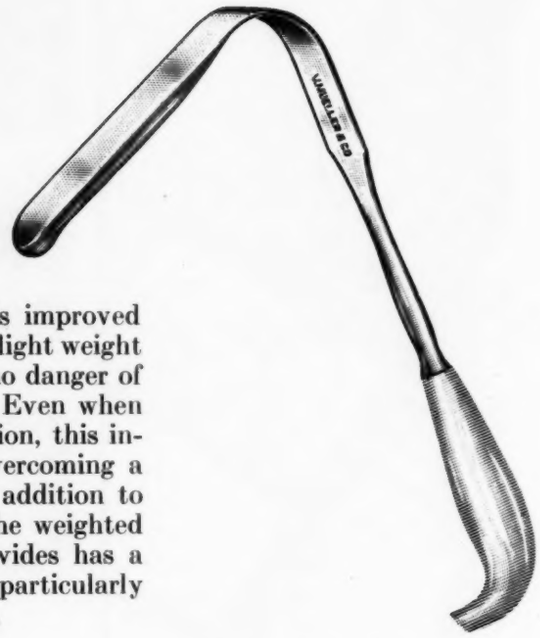

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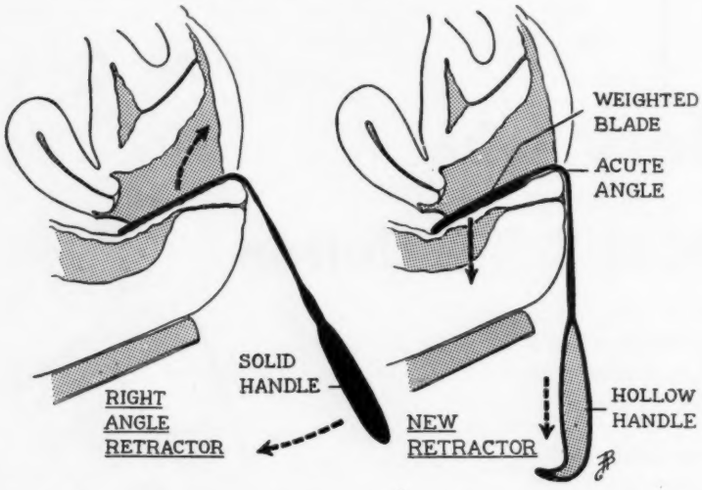

The sketches at left, by direct comparison, show the advantages of the improved instrument. As gravity swings the weighted handle of the usual retractor toward the vertical, even if care is used the instrument has a tendency to fall out. The longer, weighted blade of the Glenner retractor provides a pull directly downward; the acute angle permits the handle to hang vertically, yet prevents the retractor from falling out when touched.

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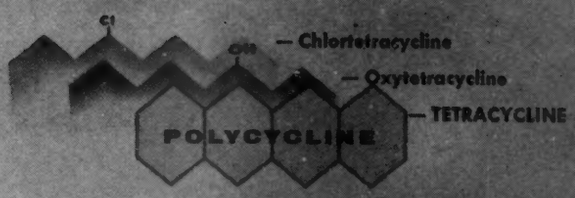

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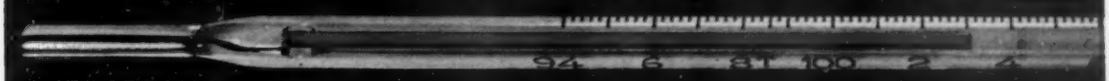

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References
(1) Kirby, W. M. M.; Waddington, W. S., & Doornink, G. M.: Antibiotics Annual, 1953-1954, New York, Medical Encyclopedia, Inc., 1953, p. 285. (2) Finland, M., & Haight, T. H.: *Arch. Int. Med.* **91**: 143, 1953.

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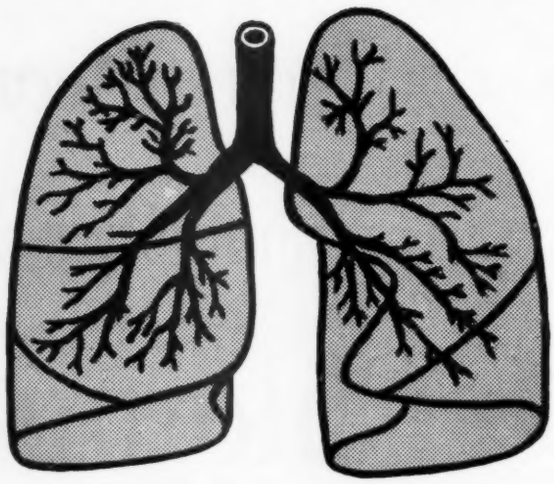

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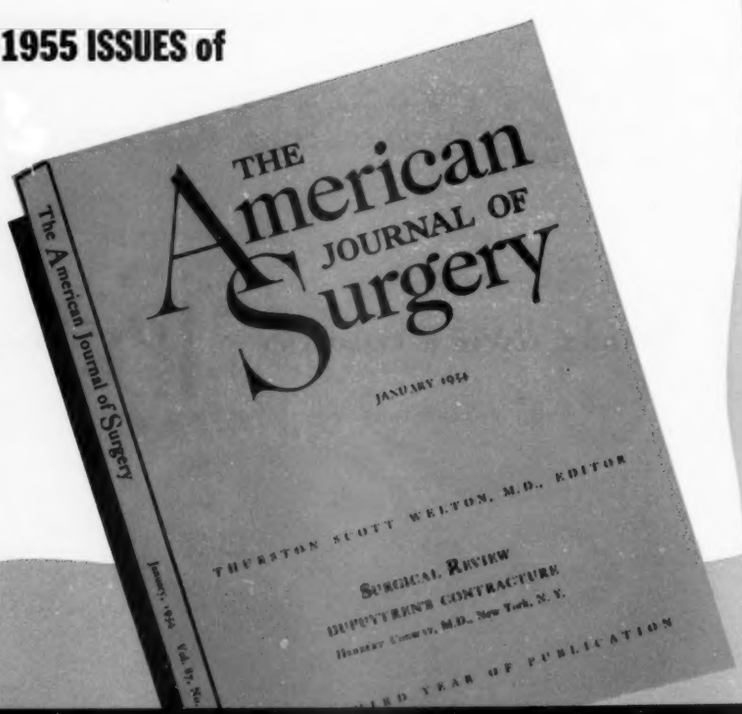

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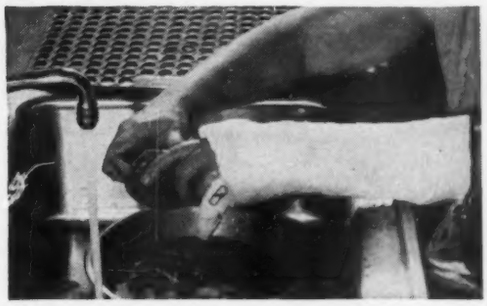

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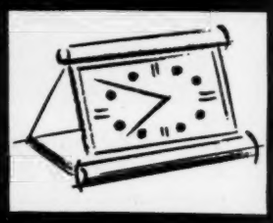

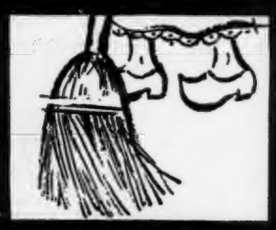

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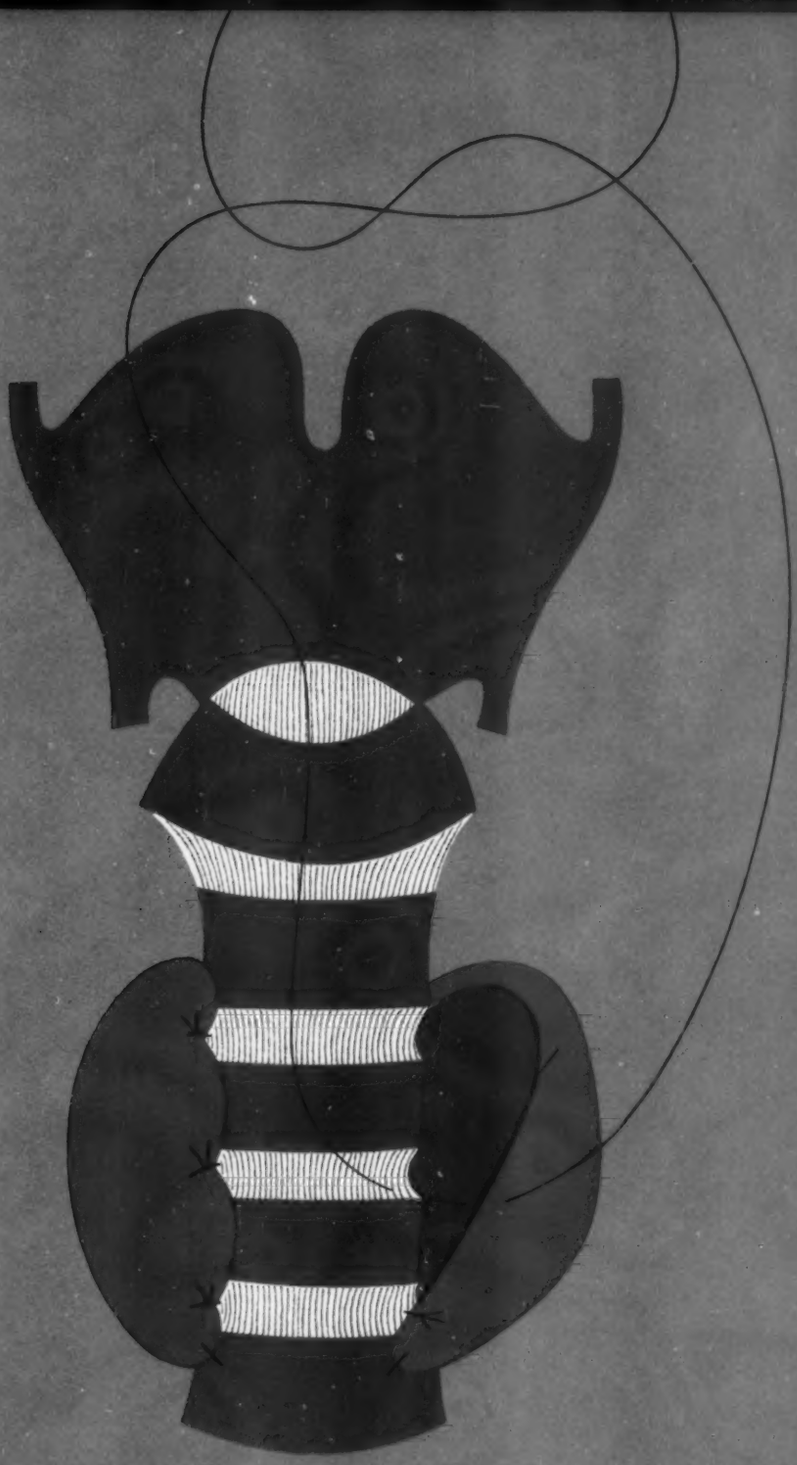

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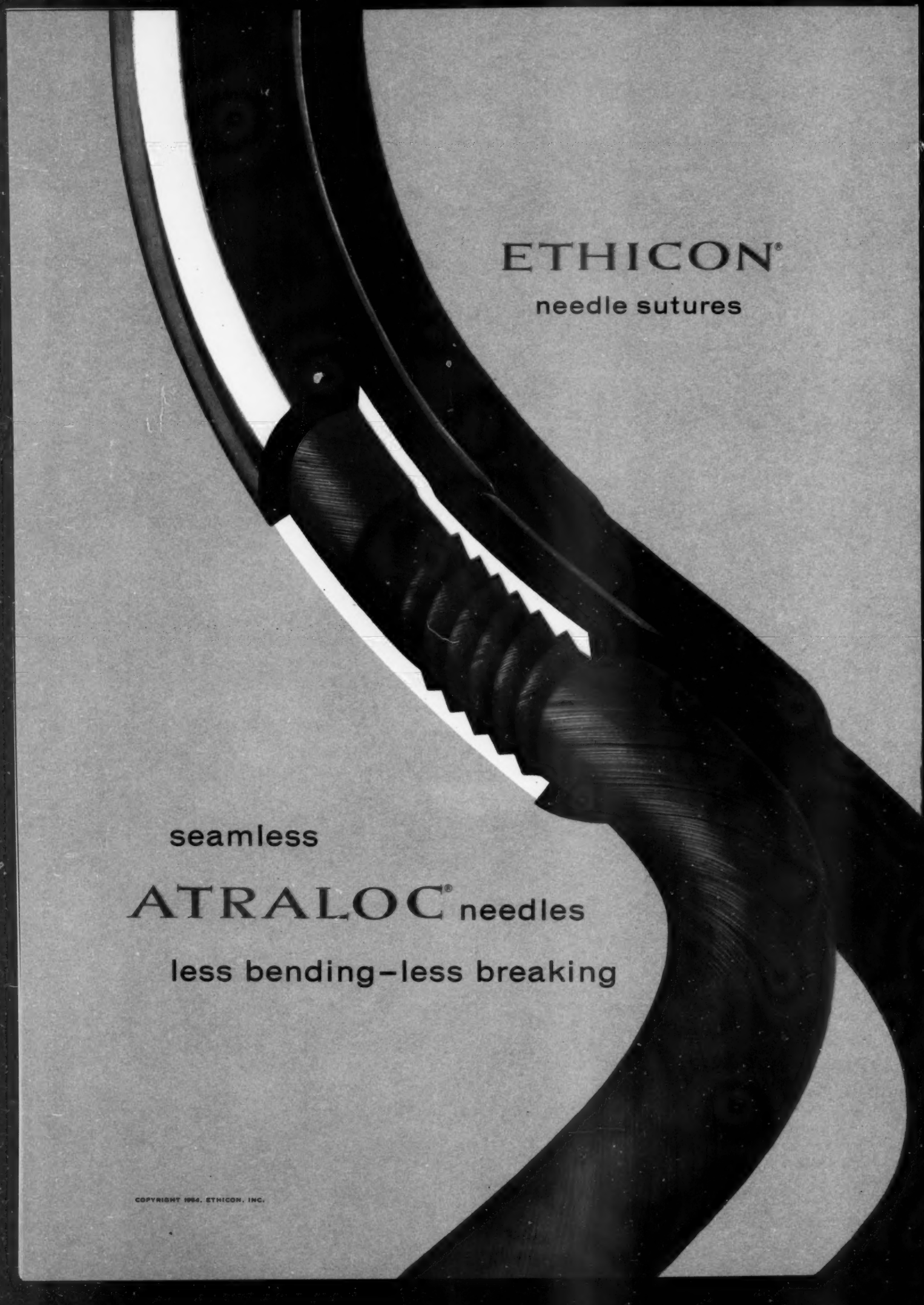

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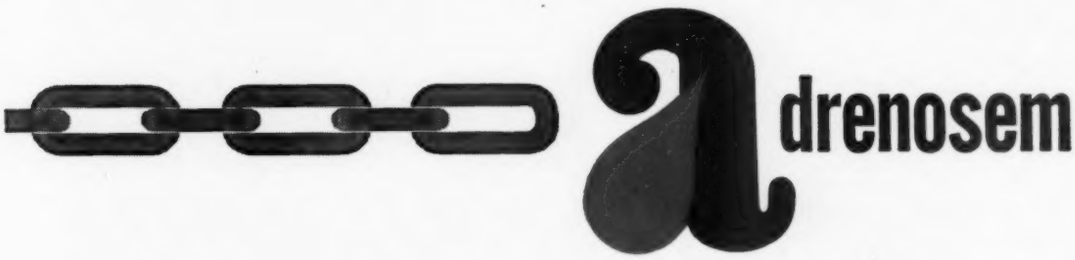
*Sherber, D. A.: The Control of Bleeding, Am. J. Surg. *86*:331 (Sept.) 1953.



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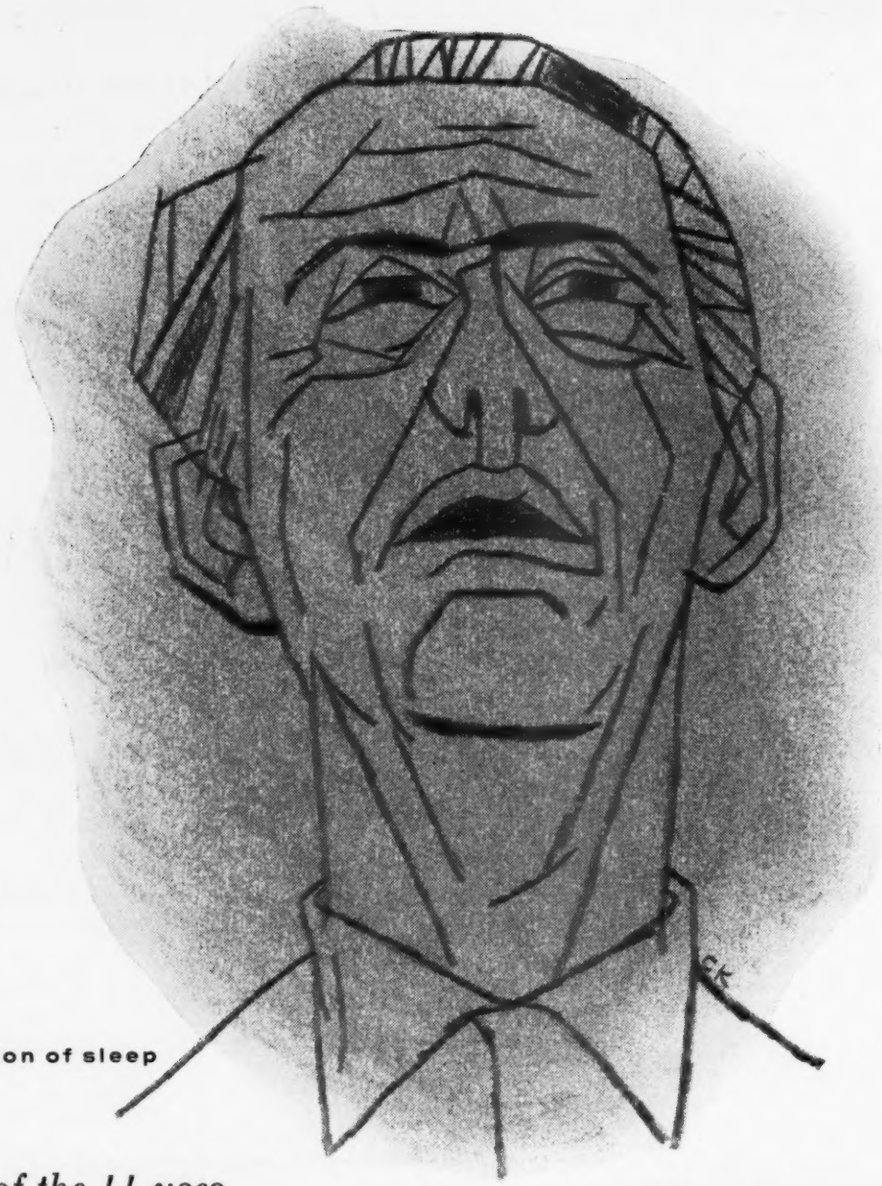

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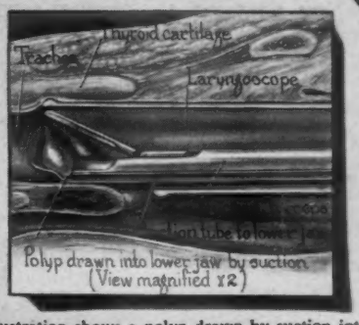


Illustration shows a polyp drawn by suction into lower jaw of Broyles laryngeal suction forceps.

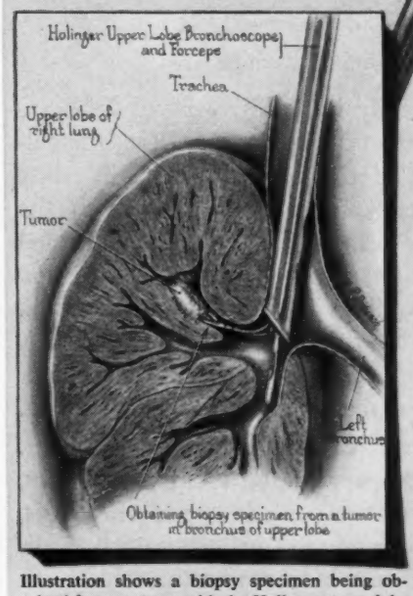
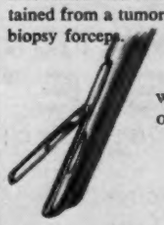
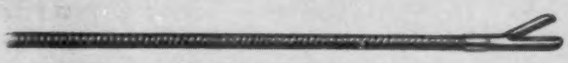

Holinger Upper Lobe Bronchoscope and Forceps
Trachea
Upper lobe of right lung
Tumor
Left bronchus
Obtaining biopsy specimen from a tumor in bronchus of upper lobe

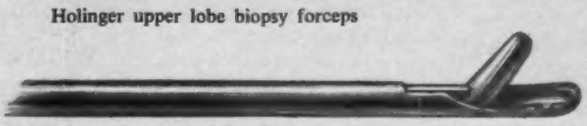
Illustration shows a biopsy specimen being obtained from a tumor with the Holinger upper lobe biopsy forceps.



Broyles laryngeal suction forceps

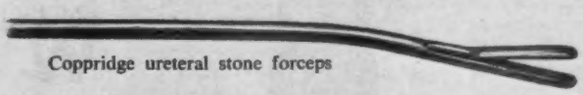


Holinger upper lobe biopsy forceps



Lowsley grasping forceps

Turell rotating rectal biopsy forceps



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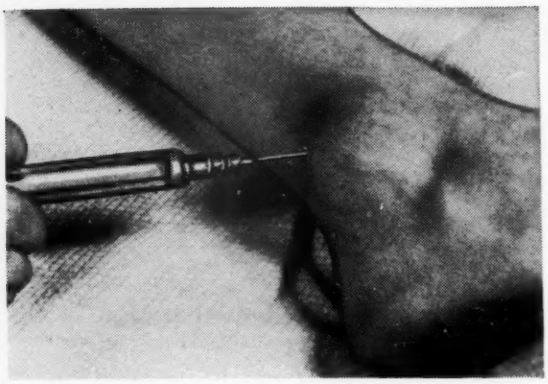

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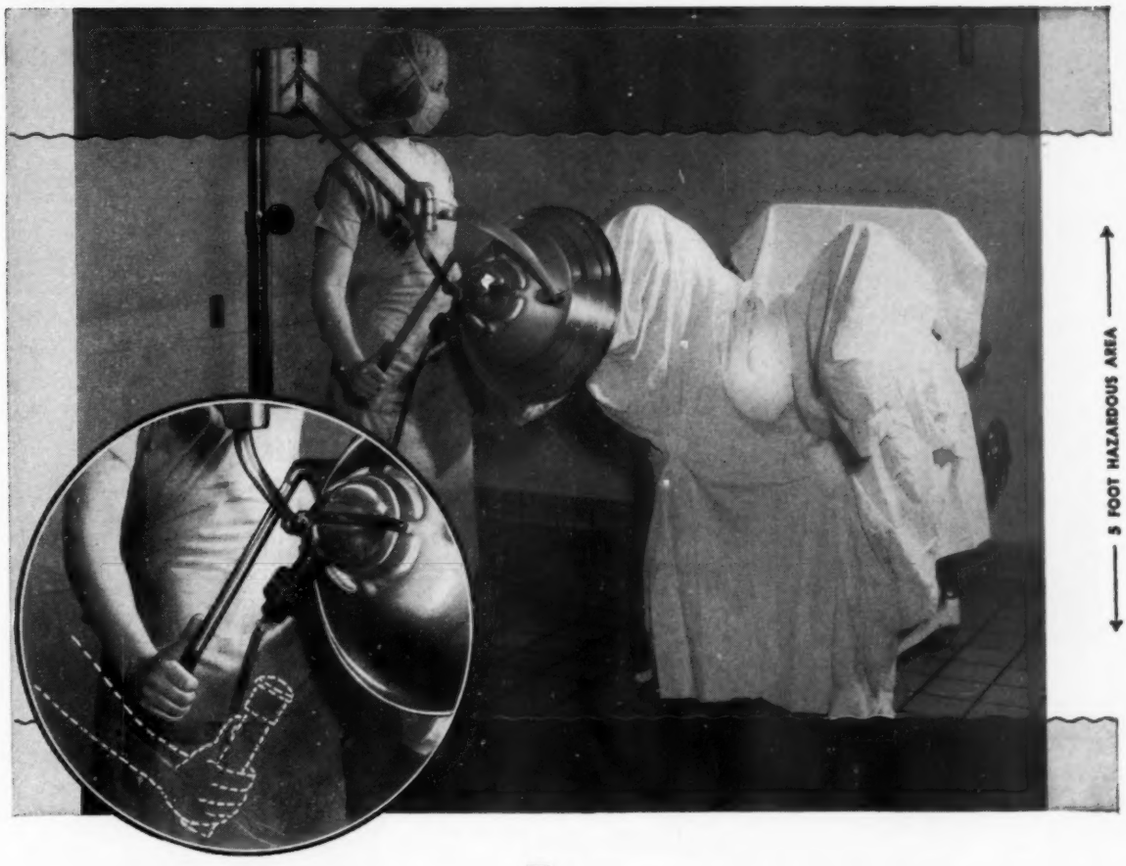

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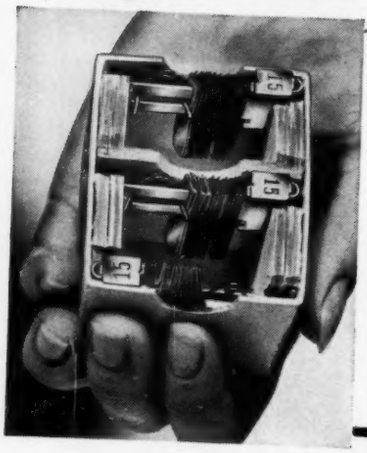

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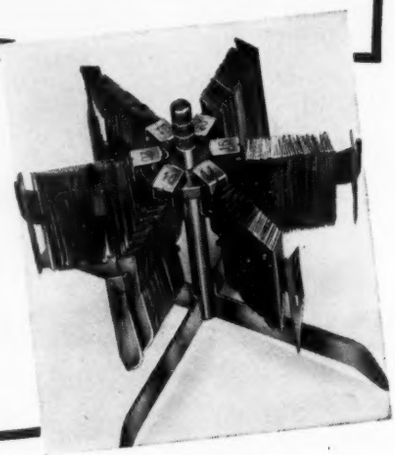

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VOL. 88 SEPTEMBER 1954 NUMBER THREE

Editorial

THE SILENT GALLSTONE SPEAKS*

In one respect things have not changed since Sir William Osler said sixty years ago, "There is no medical treatment for gallstones." It may seem strange that we should discuss this subject with a group of surgeons who are already convinced that the treatment of gallstones is surgical; yet approximately 15,000 people in the United States will die this year of gallstones or one of the associated complications. It seems likely that this appalling number might be substantially reduced if we could correlate our thinking and adopt a more rationally uniform approach to the problem.

Gallstones are never normal. They always represent disease. For purposes of this discussion it matters not too much whether the initial cause is metabolic, chemical, obstructive or infectious if we constantly remember that gallstones do represent disease. Viewed in this light, it is unfortunate that the euphonious, but nevertheless erroneous, term "silent gallstone" should ever have been invented. Is there in reality ever a completely silent (harmless) gallstone?

The gallbladder is a contractile, essentially funnel-shaped reservoir in its functioning state which, by the force of its own muscular contractions, habitually empties its contents through its narrow cystic duct spout into the common bile duct. In order for this function to proceed in a smoothly normal fashion these contents must be liquid. From a strictly mechanical standpoint the addition of sand, gravel or rocks will inevitably interfere with proper emptying. Admittedly, many times this interference might be minimal or symptomatically subclinical, but the x-ray report of "normally functioning gallbladder with stones" represents the initial wet spot on the kitchen ceiling from the small leak in the roof. The cloudburst with the plaster falling off and the room filling with water comes later—but too often it comes.

What, then, should be our attitude when gallstones are demonstrated unexpectedly, or incidentally, by x-rays or have been palpated during a laparotomy for some other condition and when there are no gallstone symptoms? Are we not derelict in our duty if we do not urge their removal?

What about the patient who has had an attack of acute obstructive cholecystitis who placidly keeps his gallstones with the sense of false security that they are causing no trouble? Are we not, as surgeons, delinquent if we do not insist on operation during the quiescent phase in order to prevent repetition of another acute attack or migration of a stone into the common duct with all the hazards of obstructive jaundice, pancreatitis or obturation of a stone in the ileum, or even carcinoma of the gallbladder?

During the seventy-five years since it was first performed as a deliberate procedure, surgical excision of the diseased but relatively

* This is a brief version of a paper given at the Southern California Chapter of the American College of Surgeons meeting, Santa Barbara, Calif., February 6, 1954.

quiescent gallbladder has indeed become a very safe and satisfactory operation. It is being performed by good surgeons all over the world with a mortality rate of about ½ of 1 per cent and a morbidity rate of almost zero. However, when we wait for acute gangrenous cholecystitis, the operation becomes ten times as dangerous with five times the risk of injury to the ducts. Hepaticoduodenostomy, hepaticojejunostomy, end-to-end common duct reconstructions and the Longmire procedure are all ingenious and heroic operations but they are poor substitutes for the normal common bile duct. I am sure that you will share with me the hope that all these operations might some day be of historical interest only.

Regarding the gallstone patient with coexistent disease, the literature is full of statements such as: "Gallstones should be removed unless contraindicated by the age of the patient or the presence of intercurrent disease." People of advanced age tolerate an elective cholecystectomy better than they will weather a single attack of acute obstructive cholecystitis. Rheumatic and coronary heart disease have too frequently been used as excuses against proper surgical treatment of gallstones. Again, it has been repeatedly shown that the patient with a handicapped heart can usually be prepared to withstand planned anesthesia and operation with an ultimate calculated risk considerably less than that which accompanies acute cholecystitis or obstructive jaundice with probable, much more serious emergency operation becoming mandatory with little or no opportunity for medical preparation. How many coronary attacks have been in reality gallstone colics? How many anginal spasms have been precipitated by the fear and dread of recurrent biliary colic? Those of us who might have had gallstone colic will be the first to agree that such fearful anticipation of pain is not the best prophylactic against a coronary attack. Elective cholecystectomy can be performed today on diabetic patients with a safety comparable to that in a non-diabetic patient. Acute cholecystitis or obstructive jaundice, however, may easily get the patient so out of balance as to increase the hazard of surgery markedly. Then, too, the removal of an infected gallbladder with stones often actually improves the patient's diabetes. These are just a few of the reasons why we might reverse some of our previous concepts and quite logically say that heart disease, diabetes, advanced age and other coexistent handicaps might often be an argument for, rather than against, the elective removal of temporarily silent gallstones.

We know, of course, that all people with gallstones do not develop serious complications. We are equally aware of the fact that many do not even have biliary colics or other recognizable symptoms of biliary disease. Some will present acute cholecystitis or obstructive jaundice as the first symptom of the presence of gallstones. The mortality figures for operations on the biliary tract reveal the fact that most of the deaths are in the complicated groups of acute cholecystitis, obstructive jaundice, cholecystoenteric fistula, gallstone ileus and carcinoma of the gallbladder. If operations for definitely demonstrated gallstones could be performed before the onset of these dangerous complications, results would be proportionately better.

There is no such thing as a completely silent gallstone. Gallstones are not only indications of disease but also are always potential causes of complications far more dangerous than is simple elective cholecystectomy performed during a quiescent phase. Coexisting disease should often be an indication, rather than a contraindication, for cholecystectomy. General acceptance of this attitude toward calculous cholecystic disease by the general practitioner, internist, surgeon and patient will not only lead to a greater degree of satisfaction to patient and doctor alike but also will save many lives. This is a responsibility we are obliged to accept.

JAMES M. MARSHALL, M.D.,
Diplomate, American Board of Surgery,
Pasadena, California

Necrosis in Rectal Carcinoma Following Intravenous Chemotherapy

LAURENCE G. BODKIN, M.D., *Diplomate, American Board of Proctology, Brooklyn, New York*

Read at Staff Conference, St. Mary's Hospital, Brooklyn, New York, on February 25, 1954.

IN a small group of cases partial necrosis of carcinomatous tissue has been observed shortly after intravenous administration of two chemicals. An inert vehicle carrying with it an active agent has been used. The therapy has been based upon physical and chemical theories of mine regarding mechanisms involved in polypoid growth. The treatment is applicable in its present state only to late inoperable cases of rectal cancer.

A rectal polyp has always seemed to me to represent a small imperfect hydraulic pump. Its formation may be influenced by two basic rules of physics, Pascal's law and Bernoulli's principle. Figures 1 to 3 illustrate the comparison between the hydraulic pump and the rectal polyp.

Pascal's law states that pressure on a contained liquid is transmitted equally in all directions. For example, a liquid entering through a channel 1 inch square with a pressure of 10 pounds to the square inch will exert a pressure of 10 pounds on each square inch of the enclosed container.

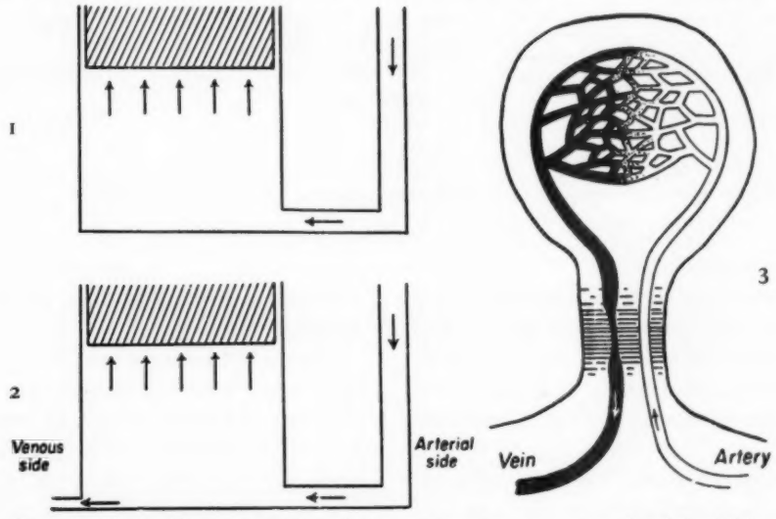


FIG. 1. Hydraulic pump. Pascal's law: pressure on a contained liquid is transmitted equally in all directions.

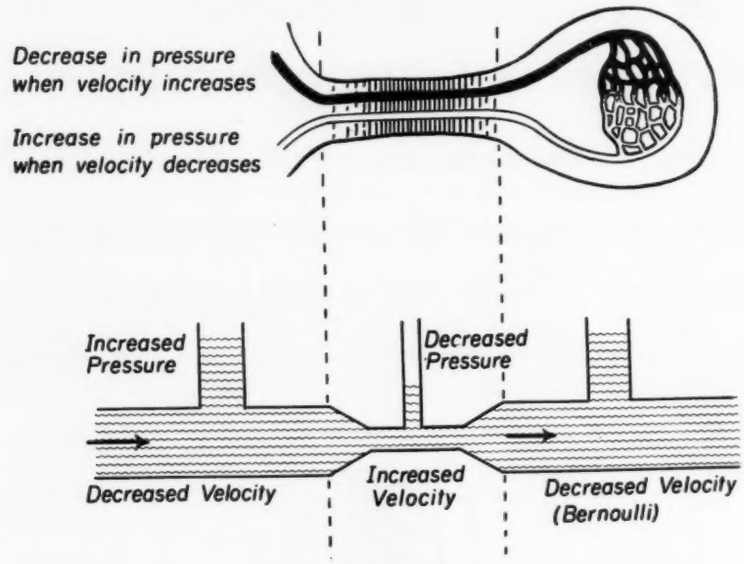
FIG. 2. Imperfect hydraulic pump, with outflow.

FIG. 3. Formation of imperfect hydraulic pump (vein compressed more than the artery).

If even an imperfect hydraulic pump is formed in a rectal polyp, considerable pressure will be exerted within the head of this structure. Enlargement will be aided and the tissues within may even be supplied with chemical nutrients under pressure.

This mechanism, if it exists, may be influenced by factors present in a structure of

polypoid form. Bernoulli's principle states that there is a decrease in pressure when the velocity increases and an increase in pressure when the velocity decreases. The first condition may be present in the stalk and the second in the tip of the polyp. Figures 4 and 5 demonstrate this point. Systole and diastole may further affect local pressure changes and elasticity of the patients' tissues may be a factor. Incidentally, most of the patients studied in the past four years have had an unusually elastic, smooth skin.[1]



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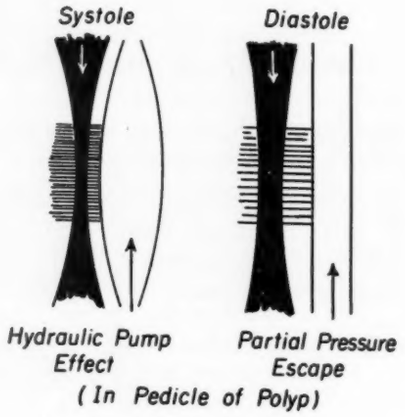

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FIG. 4. Bernoulli's principle as applied to polypoid structure.

FIG. 5. Pedicle of polyp during systole and diastole.

With the aforementioned as a working hypothesis, response to intravenous therapy has been studied. It has been noted that the surface of a rectal carcinoma is a mass of small polypoid structures. Furthermore, fluid in the form of plasma or blood is constantly escaping from it. Except for the kidneys fluid does not escape as readily from other sites in the body. A large carcinoma of the rectum represents a surface from which there is marked leakage of fluid.

In cases of shock or blood loss an inert chemical or plasma expander is often used and it is only slowly eliminated from the body. It may even remain in the blood stream for several weeks. Small amounts escape from the kidney; very little elsewhere. I believe that when it is administered to a patient with rectal carcinoma it is excreted slowly through the carcinomatous tissue. I also believe that it can be employed as a vehicle for active chemical agents and that it will cause the same to be slowly discharged through the cancer mass. These active chemicals are attached to the heavy molecules of the plasma expander by adsorption.

Many vehicles and a large number of chemicals were used in studying a series of thirty patients. The results can be briefly summarized. The most effective vehicle or plasma substitute used so far has been polyvinylpyrrolidone (PVP or vinisil®).* In order to detect its presence in the carcinoma various dyes were added to it. In eleven cases indigo carmine was found to be a fairly simple and effective "tracer." I was quite surprised to find that this dye acted not only as a "tracer" but also as a local irritant to the carcinomatous tissue when used in high concentration.

Indigo carmine is a dye that has been used in routine tests of kidney function. Five cubic centimeters of an .8 per cent solution are employed when making such a test. In this study, however, the dosage has been increased to ten times this amount. Great reliance has

* Vinisil supplied by Dr. Arnold E. Osterberg of Abbott Laboratories, Chicago, Ill.

been placed upon the adsorptive powers of the heavy molecular vehicle.

Following the intravenous administration of PVP and indigo carmine, both gross and microscopic changes in the carcinomatous tissue have been consistently observed in this small series of cases. As a rule, three to five weeks elapsed before any changes were noted.

The gross changes are those of partial necrosis with whitened tissue often containing some slight blue tinge. At times the blue is present in dark spots that are easily distinguishable. Microscopic findings are those of necrosis, inflammation, colloid degeneration and vacuolization of the nuclei of the cancer cells, in greater or lesser degree. The gross changes are easily observed and in some instances have been photographed. A fairly large number of biopsies have been done.

In the group to be discussed there were eleven cases. Each patient was given 500 cc. of PVP and 50 cc. of indigo carmine intravenously. Two to five injections were given at intervals of two to seven days. One patient, who received only two injections of this particular combination, has shown little or no reaction. Another patient has responded sluggishly. The lesions of the others have shown definite necrosis.

The question naturally arises regarding the

TABLE 1

Patient	Age (Yr.)	Surgery	Number of Injections (Intravenous)	Pathologic Changes	Gross Changes	Living or Dead
W. M.	59	Colostomy	20 in past year	Suppurative necrosis; colloidal degeneration	Considerable white necrotic tissue; occasional slight blue discoloration	L.
A. P.	54	Abdominoperineal resection; ileotrans colostomy for recurrence	4	Degeneration, necrosis; some tumor cells show marks of nuclear vacuolation; granulation tissue areas	Partial necrosis and dark blue tissues; areas of soft "granulation tissue"	L.
F. S.	63	Colostomy, March, 1950	20	Marked suppurative necrosis	Partial necrosis and blue discoloration	L.
W. S.	50	Colostomy, December, 1953	4	Chronic inflammatory exudate; edema degenerate; necrosis	Partial necrosis and blue discoloration	L.
E. S.	75	Colostomy	7	Inflammatory exudate; necrosis; hemorrhage	Areas of necrosis and discoloration	D. (autopsy)
A. S.	79	Colostomy, 1952	8	Partially necrotic and suppurative tissue; other tissues, well preserved carcinoma	Few areas of necrosis; other areas typical carcinoma, yet not advancing	L.
M. H.	64	Perineal extirpation	4	Considerable inflammatory reaction, but necrosis moderate or absent	Lesion smaller; few areas of necrosis seen	L.
J. R.	54	Colostomy	4	Necrotic tissue containing remnant of carcinomatous tissue, secondary fibrotic changes	Areas of necrosis with white-blue tissues seen	L.
J. A.	74	Cauterization; no colostomy	2	Minimal necrosis	Lesion practically stationary; no necrosis seen	L.
O. J.	83	Colostomy	4	Marked necrosis and degeneration	Marked necrosis with areas of blue discoloration seen	L.
N. L.	59	Colostomy; refused abdominoperineal	5	Considerable necrosis and degeneration	White necrotic tissue throughout central portion of lesion	L.

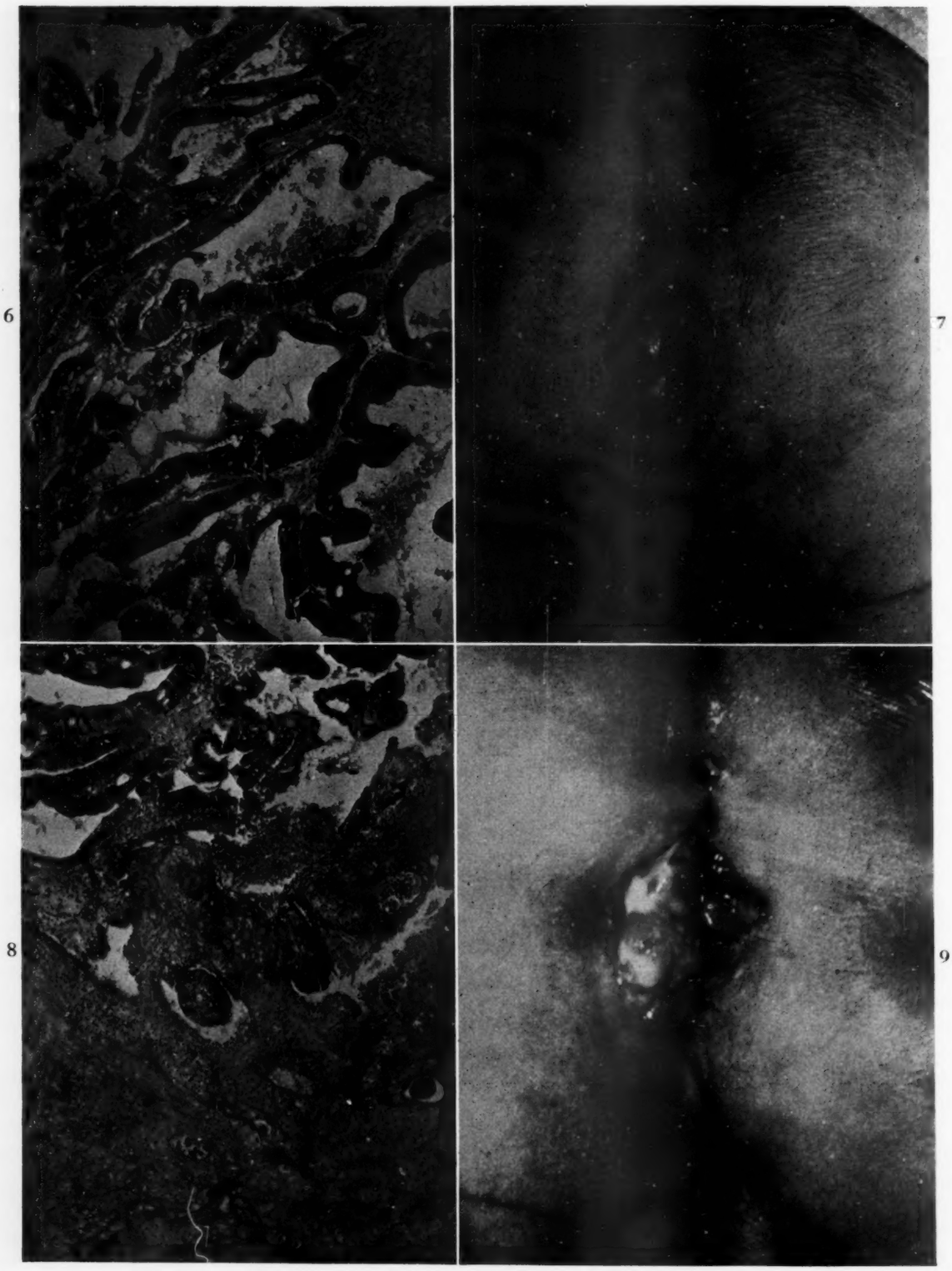

Fig. 6. Patient A. P. Adenocarcinoma recurrence in perineal scar.

Fig. 7. Superficial necrosis of carcinoma in perineal scar.

Fig. 8. Degeneration and edema of tissues seen in Fig. 7.

Fig. 9. Necrosis and blue discoloration of carcinoma.

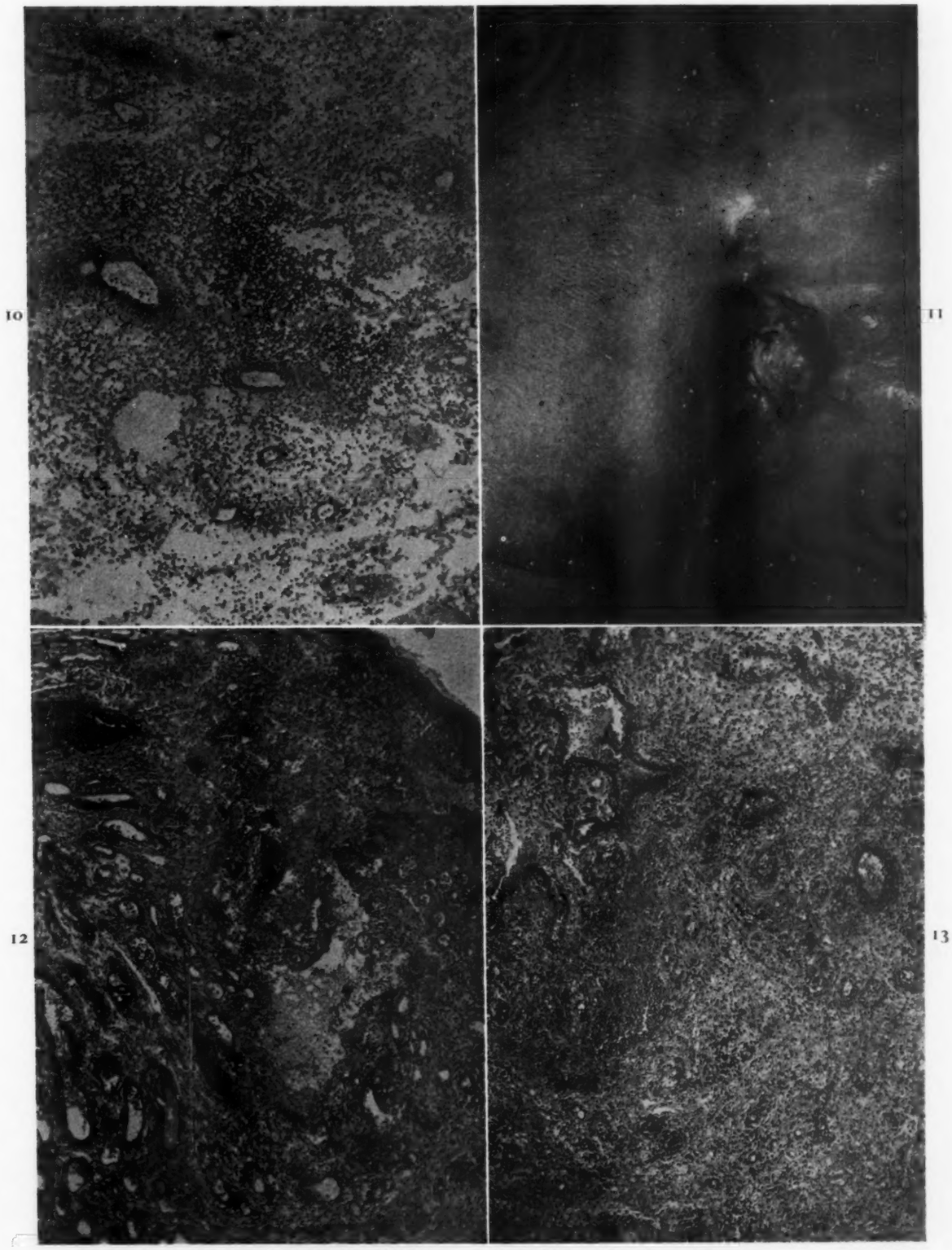

FIG. 10. Marked necrosis of tissues of same.

FIG. 11. Dark blue area on left.

FIG. 12. Edema and necrosis in same.

FIG. 13. Carcinoma tissue, one month later.

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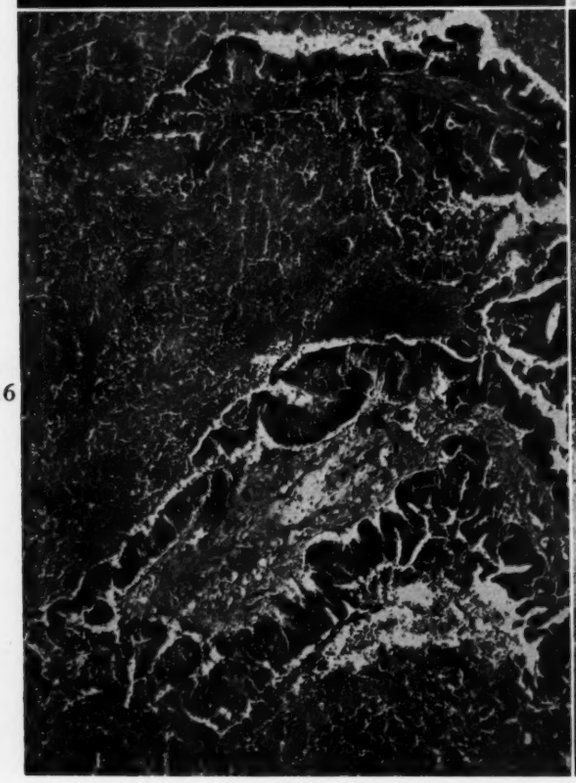

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FIG. 14. Patient F. S. Rectal carcinoma protruding from anal canal.

FIG. 15. Invasive malignant tissue from same.

FIG. 16. Necrosis following intravenous therapy.

FIG. 17. Necrosis and blue discoloration of carcinoma.

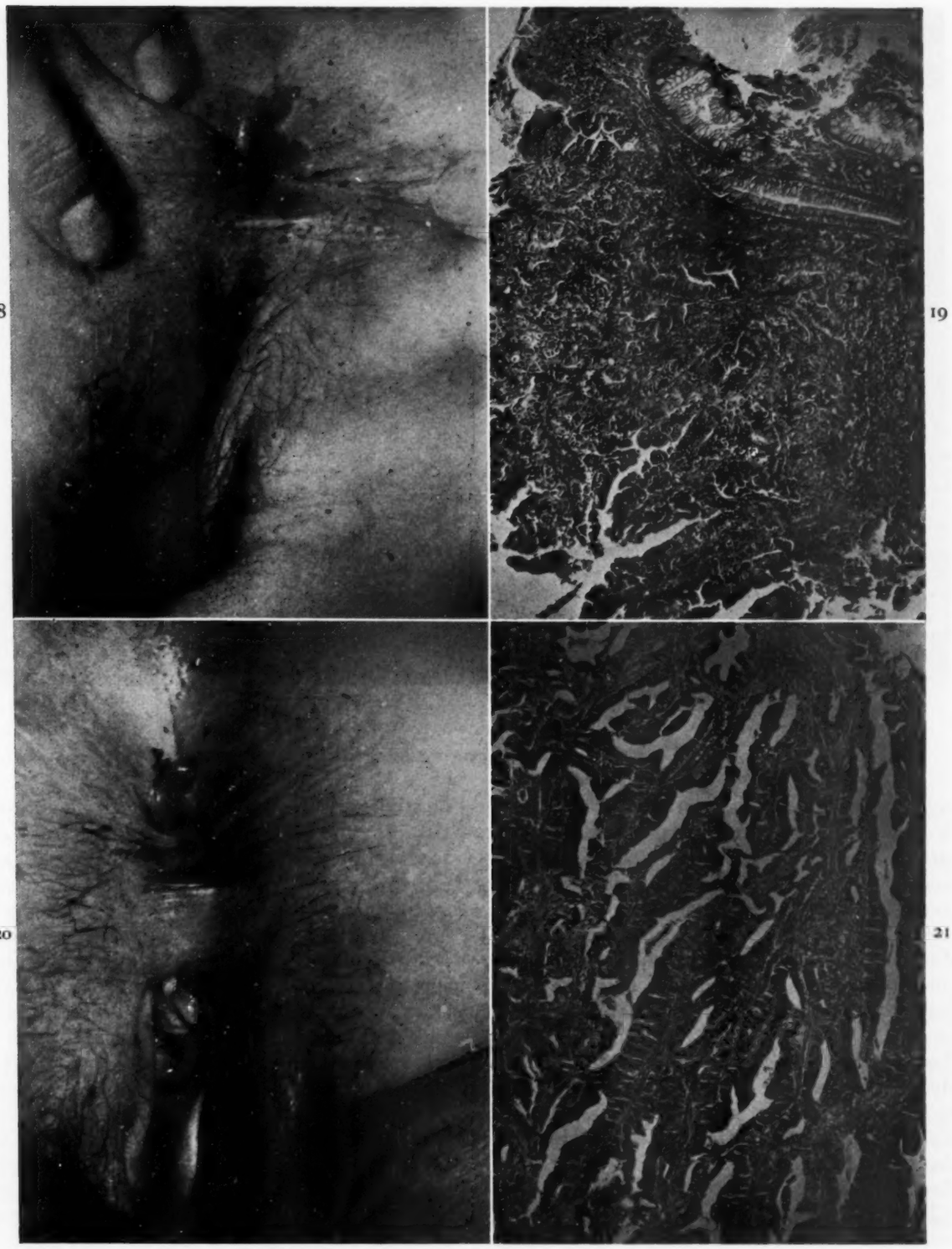

FIG. 18. Patient J. A. Rectal carcinoma extending into perianal skin.

FIG. 19. Adenocarcinomatous tissue from same.

FIG. 20. Carcinoma two months later, unchanged.

FIG. 21. Tissues unchanged by therapy, yet not advancing.

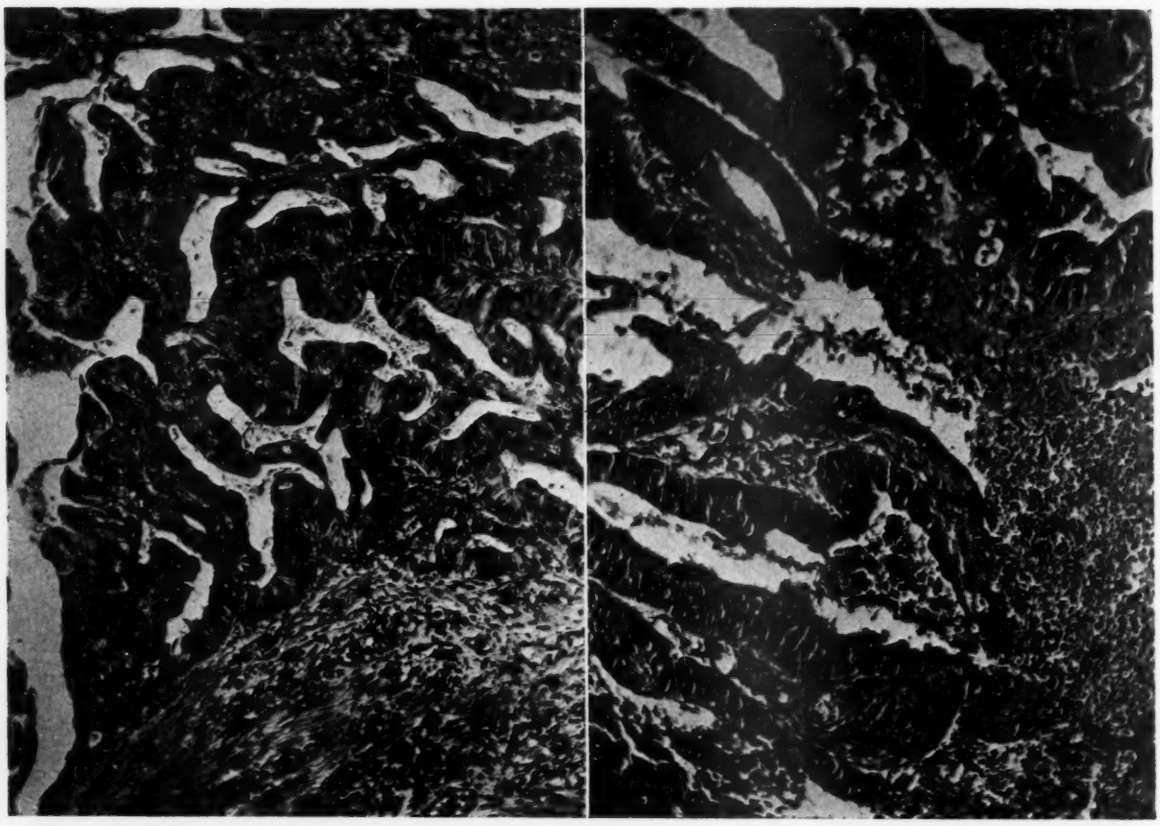

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FIG. 22. Patient W. S. Typical adenocarcinoma of rectum.

FIG. 23. Later, inflammatory exudate and partial degeneration.

fact that some necrosis is present in most carcinomas of the rectum. For purposes of comparison biopsies were done before intravenous therapy was given. The pathologists were careful to note presence or absence of necrosis and inflammation or unusual staining qualities of the carcinoma cells before and after medication. Although error cannot be avoided, some degree of comparison can be obtained by this method. Dr. William Moitrier at St. Mary's Hospital and Dr. Edward Marino at Peck Memorial Hospital reported similar findings on a large number of specimens. Recently Dr. Max Wachsteen at St. Catherine's Hospital also reported necrosis and markedly increased fibrosis.

The response to this rather unorthodox approach to the problem of cancer of the rectum is shown in Table 1. These patients all received the same intravenous therapy, although the number of injections varied.

In order to demonstrate the changes that have occurred the following illustrations may be studied. In Figure 6 recurrent carcinomatous tissue in the perineal scar of patient A. P. is seen. Highly anaplastic epithelial cells are present in a typical adenocarcinoma. Vinisil and indigo carmine were given and three weeks later a photograph showed some superficial necrosis, of no significance. (Fig. 7.) However, a specimen of tissue taken from deep within the mass was reported as showing "degeneration and edema distinctly more marked than in previous specimen taken October 9, 1953." (Fig. 8.) On November 3rd a photograph demonstrated not only areas of necrosis but also some definite blue discoloration. (Fig. 9.) Figure 10 shows marked degeneration of the carcinomatous tissues. On November 17th specimens were removed from two different portions of the growth by Dr. Moitrier. A large reddish white projection of carcinoma on the right and a small dark blue area on the left were seen (Fig. 11.) A photomicrograph of the large portion shows edema and necrosis, with nuclear vacuolation of the malignant cells.

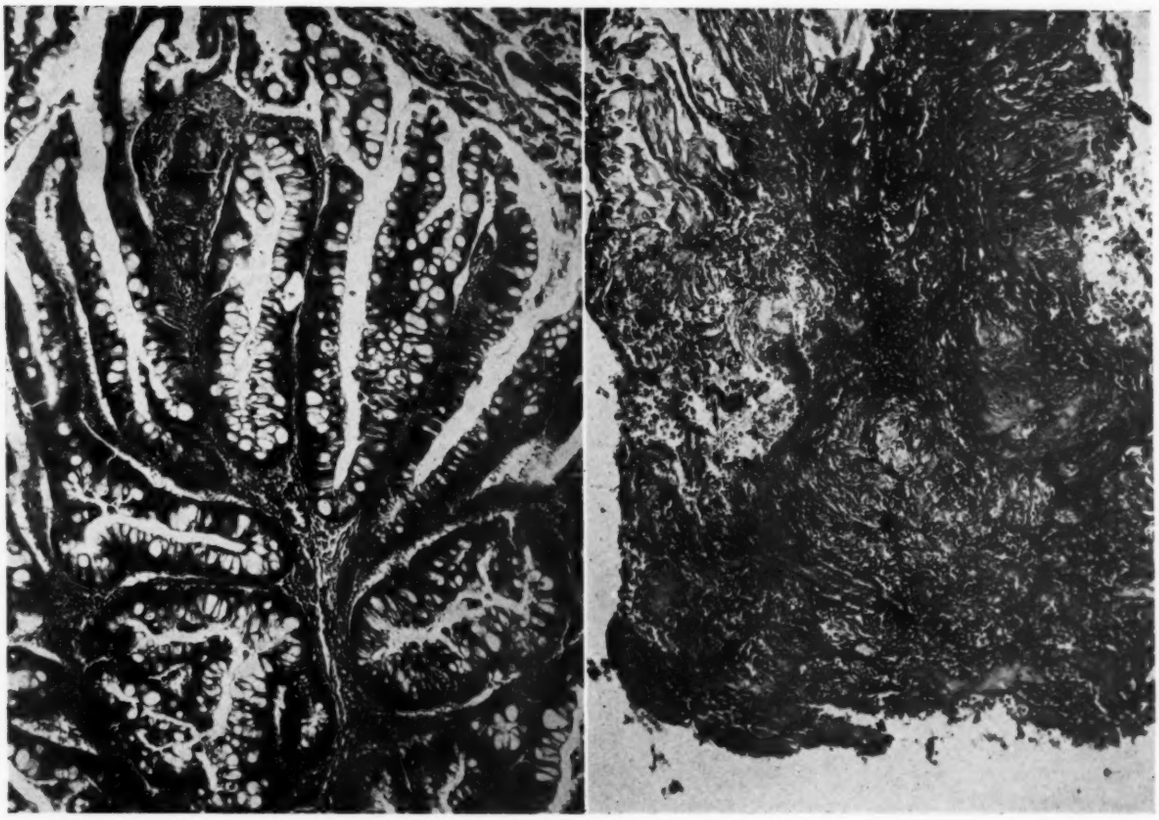

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FIG. 24. Patient W. M. Colloidal adenocarcinoma of rectum.

FIG. 25. Necrosis and degeneration following therapy.

(Fig. 12.) Tissue from the dark blue area showed granulation tissue and degeneration but no definite malignant cells. Biopsy was done again in the same manner on December 11th and showed the same conditions. (Fig. 13.) In the portion containing so much granulation tissue and degeneration a few faint carcinoma cells were found, removing any doubt that this was originally carcinoma tissue.

Another patient, F. S., has been under my care for some time. In April, 1953, his carcinoma began to protrude from the anal canal. (Fig. 14.) A typical invasive malignant neoplasm of adenocarcinomatous structure was present, with no colloid production or necrosis except for a few pinpoint areas. (Fig. 15.) This carcinoma had always proved resistant to any form of intravenous therapy although such therapy had been administered to the patient several times. The concentration of indigo carmine was gradually increased, and on October 28th marked suppurative necrosis was found. (Fig. 16.) In November, 1953, necrosis and blue discoloration were seen in the then greatly enlarged carcinomatous mass. (Fig. 17.)

One patient, J. A., has shown only negligible changes so far. Figure 18 shows the condition on October 8, 1953, with the lesion extending outward onto the perianal skin. Figure 19 shows the usual adenocarcinomatous structure. On December 10, 1953, there was little change. (Figs. 20 and 21.) This patient has received only two intravenous injections containing the large dosage of indigo carmine. Subsequent injections may change the picture. It is interesting to note that the lumen has never closed completely and that colostomy has never been performed, even though it was indicated more than a year ago.

Photomicrographs in other cases before and after treatment may be briefly reviewed. Figure 22 shows the typical adenocarcinomatous structure in patient W. S., admitted to St. Mary's Hospital on November 21, 1953. Figure 23 shows chronic inflammatory exudate, hemorrhage, degeneration and necrosis.

26 27

FIG. 26. Patient J. R. Papillary adenocarcinoma of rectum.

FIG. 27. Necrotic tissue following intravenous therapy.

Patient W. M., admitted to Peck Memorial Hospital on February 27, 1953, had carcinomatous tissue with abundant colloidal activity and minimal degeneration. (Fig. 24.) Many biopsies have been done during the past year, most of them showing marked colloid degeneration and necrosis with comparatively few areas of intact malignant cells, as in Figure 25. This carcinoma has remained almost stationary since December, 1952. The patient goes to his office each day and has never used sedatives.

Figure 26 (patient J. R.) shows a papillary adenocarcinoma. A photograph from St. Catherine's Hospital where colostomy was performed shows necrotic tissue with remnants of adenocarcinoma. (Fig. 27.) Slides of other patients in this series, before and after therapy, demonstrate similar changes.

Many other chemicals, including colloidal sulfur, arsenic, teropterin® and nitrogen mustard, have been tested. The effect upon the rectal lesions will be reported at a later date.

SUMMARY

Both gross and microscopic changes have been observed in late carcinomas of the rectum shortly after the intravenous administration of polyvinylpyrrolidone and indigo carmine. In ten of eleven cases this reaction has occurred. The treatment is based upon a theory held by the author regarding mechanisms that exist within a polypoid carcinomatous growth. Its clinical value at present is slight except for palliation and the possibility of avoiding colostomy in selected cases. Its main interest lies in the fact that this unusual phenomenon occurs in a rectal lesion after intravenous therapy consisting of a viscous, inert vehicle and an active chemical agent. A new approach to the problem of late rectal cancer is suggested based upon principles of physics and chemistry. Further clinical observations are being made and will be reported at a later date.

ADDENDUM

Patient F. S. died June 20, 1954. At complete autopsy no metastasis was found in any organ. A large carcinomatous mass of rectum involving half of both buttocks and base of scrotum showed partial necrosis throughout. The entire tumor was easily shelled out as though encapsulated. There were a few adhesions in the pelvis. There were no involved lymph glands. Necrosis was absent in other organs.

Theoretically, the entire cancer could have been removed. Several chemical compounds had been employed using vinisil as a vehicle. It is believed that the indigo carmine in vinisil followed by oxalic acid in vinisil produced this extensive degeneration of the carcinomatous mass without damage to other organs.

Observation and treatment of this case covered a period of four years.

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Working experimentally on dogs, Schatten et al. show that experimental appendiceal peritonitis is best treated by the intraperitoneal instillation of terramycin. They used concentrations of 2 mg. per ml. Ultimate survival was about 50 per cent. The technic seems a good one to try on humans found with generalized peritonitis at time of initial surgery. Surprisingly, all of the dogs treated by intravenous terramycin died, as did all the control dogs, and only half of the intraperitoneally treated animals survived. (*Richard A. Leonardo, M.D.*)

Adenomatous Polyps of the Rectum and Distal Sigmoid

A STATISTICAL ANALYSIS OF 500 PATIENTS WITH POLYPS

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As far back as 1881, when Woodward[1] first distinguished between true or primary polyps and pseudo or secondary polyps, the subject of polyps of the rectum has had popular appeal for medical authors. Although the literature on the subject of polyps of the large bowel has been voluminous in the past two decades, the subject certainly has not been exhausted. From continuing study of adenomatous polyps of the rectum, much insight has been gained into the problem of the pathogenesis of adenocarcinoma of the rectum and colon. Among others, Fitzgibbon and Rankin,[11] Bacon and Broad,[2] and more recently, Jackman and Mayo[15] have demonstrated the adenocarcinoma sequence in cancer of the colon. This article will discuss and compare incidence of polyps, relation of male to female patients, incidence of solitary and multiple polyps, incidence of sessile and pedunculated polyps, size of polyps, statistics on the level and position of polyps in relation to the circumference of the bowel, relative frequency of adenoma and villous adenoma, histologic classification and follow-up.

The study is based on a review of the records of 500 patients with polyps who were seen by the proctology department of the Milwaukee County General Hospital from January, 1947 through April, 1952. A large portion of these 500 patients had sigmoidoscopic examinations as a routine procedure prior to barium enema study, while the remainder were seen because of various proctologic complaints.

A total of 5,072 sigmoidoscopic examinations were done in finding the 500 patients who had polyps. This gives an incidence percentage of 9.85, which compares favorably with the figure obtained by several of the writers who have computed this variable (Table 1) from sigmoidoscopic examination. Only Green[12] and Christianson and Tenner[5] found a higher percentage, but none was remarkably higher.

TABLE 1

Author	Year	Patients Examined	Per cent Incidence	Average Age
Miller and Day[18]	1950	7,494	6.4	....
Hauch et al.[13]	1950	1,919	8.1	....
Turell[28]	1948	386 (under 45)	2	....
Turell[28]	1948	386 (over 45 with bleeding)	7	....
Scarborough and Klein[23]	1948	10,000	4.6	....
Diamond[10]	1952	5,980	6.81	....
Castro et al.[4]	1951	12,000	2.4	....
Colvert and Brown[6]	1948	10,200	2.3	....
Steele and Brown[24]	1949	1,500	5.2	....
Christianson and Tenner[5]	1951	2,226	12.3	55.7
Young[32]	1951	500	8.8	....
Ortmayer and Connelly[19]	1949	1,014 (well women)	1.8	43
Martin[16]	1940	1,693	4.43	45.3
Deddish[8]	1948	1,011	6.0	....
Green[12]	1941	640	10.8	....
Burnikel and Schutte	1954	5,075	9.85	64.7

Several were considerably lower. Castro, Ault and Smith,[4] Colvert and Brown,[6] and Scarborough and Klein,[23] reporting on a large series of examinations, found a much lower incidence percentage. The average age of the patients examined is not given in the three last reports mentioned. The average age of the patients in this study was 64.7 years.

In the group of 500 patients there were 324 white males, 160 white females, thirteen Negro males and three Negro females. This gives a total male finding of 337 as compared to a total female finding of 163, or roughly, two to one male over female, as has been reported by

Miller and Day,[18] and also by Hauch, Buie, Bargen and Smith.[13] Scarborough and Klein[23] found the incidence in males only 10 per cent greater than the incidence in females, while in the study of Sandusky and Parsons[22] 53 per cent were males and 47 per cent females.

TABLE II

Number of polyps	1	2	3	4	5	Over 5
Number of patients	372	75	27	9	7	10
Percentage	74.4	15	5.4	1.8	1.4	2

TABLE III

Size of polyps	1 to 3 mm.	3 to 5 mm.	5 mm. to 1 cm.	1 to 2 cm.	2 to 3 cm.	Over 3 cm.
Number	369	157	72	72	28	6
Percentage	52.4	22.3	10.2	10.2	3.9	0.8

It was not determined if equal numbers of male and female patients were examined to find the 500 patients with polyps.

The study shows 74.4 per cent of the polyps to be solitary. (Table II.) Young[32] found that 81.8 per cent of the polyps in his series were solitary. Several authors have found percentages of single polyps in the 60's. Those include Swinton and Warren[26] 65 per cent solitary, Demuth, Cherney and Fitts[9] 67 per cent solitary, Sandusky and Parsons[22] 63.8 per cent solitary, and Scarborough and Klein[23] 64 per cent solitary.

In the group of 500 patients there were 569 sessile polyps and 135 pedunculated polyps. Ravdin and Ravdin[21] make the statement that most polyps are pedunculated and few are truly sessile. Our findings would disagree with this statement.

The distribution as to size is shown in Table III. This follows very closely the size distribution spread found by Young.[32] As one would expect, about half of the polyps seen were from 1 to 3 mm. in size, but it is against these smaller polyps that the best prophylactic and preventative blows can be struck. A polyp of this size can easily be destroyed by fulguration as an office procedure. It is our policy to biopsy all polyps, no matter what the size. During one of our teaching clinics two small 1 to 3 mm. polyps on the anterior wall were noted during routine sigmoidoscopy. Believing that this finding presented an opportunity to demonstrate to the students the technic of performing a rectal biopsy, a biopsy was done. The pathologist reported carcinoid of the rectum. Weingarten and Turell[30] report a small 2.5 mm. polyp that underwent malignant change in a six-year time lapse, and they emphasize the importance of removal and histologic study of all polyps or excrescences. Perhaps fulguration would have destroyed both those lesions, but certainly we can be more secure in our postoperative observation of those patients if we have had the benefit of histologic study of every lesion destroyed.

TABLE IV

Level in inches	1	2	3	4	5	6	7	8	9	10
Number of polyps	35	54	67	104	166	123	88	41	10	4
Percentage	5.0	7.8	9.7	15.0	24.0	17.8	12.7	5.9	1.5	.6

TABLE V

Location	Number	Percentage of Total
Anterior	131	20.8
Posterior	143	22.7
Left lateral	76	12.0
Right lateral	60	9.5
Right anterior	55	8.7
Right posterior	71	11.2
Left anterior	51	8.1
Left posterior	42	6.6

From the thirty-five polyps (5 per cent) seen at the 1 inch level (Table IV) there was a gradual increase in numbers of polyps seen at the various levels until the peak was reached at 5 inches, and then there was a gradual decrease in number of polyps seen, with only four polyps (0.6 per cent) being seen at the 10 inch level. Our findings (Table IV) are consistent with those of Castro, Ault and Smith.[4] One hundred sixty-six or 24 per cent of the polyps we observed were at the 5 inch level. In their 352 polyps they also found the largest number (sixty-three) at this 5 inch level.

In describing polyps seen through the sigmoidoscope, in addition to recording the level at which noted, we also usually record the position in the circumference of the bowel. These positions are: anterior, posterior, left

lateral, right lateral, right anterior, right posterior, left anterior and left posterior. Our findings as related to position are shown in Table v. Again our findings are similar to those of Castro, Ault and Smith[4] in that there seems to be an equal distribution between those

fications will be considered as a group in a later portion of this study and were as follows: adenocarcinoma, twenty-one; adenocarcinoma, grade I, twenty-two; adenocarcinoma, grade II, nine; atypical changes, thirty-one; and early malignant changes, forty-nine. Thus in

TABLE VI

Position	1 Inch	%	2 Inches	%	3 Inches	%	4 Inches	%	5 Inches	%	6 Inches	%	7 Inches	%	8 Inches	%	9 Inches	%	10 Inches	%
Anterior.....	9	25.7	18	33 3	25	33.3	25	22.3	25	16.6	15	16.3	10	14.7	3	12	0	0	1	25
Posterior.....	1	2.8	9	16.6	13	17.3	21	18.7	35	23.4	29	31.5	21	30.8	9	36	2	50	1	25
Left lateral....	5	14.2	2	3.7	6	8.0	19	16.9	23	15.4	13	14.1	8	11.7	3	12	0	0	0	0
Right lateral...	5	14.2	9	16.6	11	14.6	13	11.6	13	8.7	4	4.3	4	5.8	1	4	1	25	0	0
Right anterior.	7	20.0	7	12.9	7	9.3	8	7.1	8	5.3	8	8.6	5	7.3	2	8	1	25	1	25
Right posterior	2	5.7	4	7.4	3	4.0	15	13.3	22	14.6	9	9.7	12	17.6	2	8	0	0	0	0
Left anterior...	4	11.4	3	5.5	7	9.3	12	10.7	9	6.0	6	6.5	3	4.4	4	16	0	0	0	0
Left posterior..	2	5.7	2	3.7	3	4.0	9	8.9	14	9.3	8	8.6	5	7.3	1	4	0	0	1	25

anterior and those posterior, with a total anterior of 237 and a total posterior of 256. The left lateral position had seventy-six or 12 per cent of the total number of polyps, while the right posterior had seventy-one or 11.2 per cent of the total. We found the least number of polyps in the left posterior position (forty-two or 6.6 per cent). At the higher levels the position of the polyp as noted through the sigmoidoscope may not be accurate, due to rotation of the bowel.

We determined the number of polyps and the percentage of the total number in the eight positions at the various levels in our effort to see if there was any trend or correlation. The results are shown in Table VI. One observation can be made from this table, namely, that at the lower levels more polyps are found in the anterior positions, while at the 5 inch level and above the majority of the polyps are in the posterior positions.

In the group of 500 patients with polyps there was a total of 704 polyps. In 377 of these cases either a biopsy portion or the entire polyp was sent to the pathology department for histologic study. A report of benign polyps was given on 226 of the specimens submitted. In the five-year period several pathologists examined the tissue so that complete uniformity of opinion was not possible. Epithelial hyperplasia was reported in three cases. A diagnosis of papillary adenoma was made in nineteen cases. Carcinoid, leiomyoma, lymphoid nodule and submucous lipoma were each reported one time. The following five classi-

our study 60 per cent of the polyps submitted to the pathologist were benign. If the sigmoidoscopist considered the polyps to be a frank adenocarcinoma, it was not included in this study. Those lesions that from the sigmoidoscopic appearance were considered possible malignancies, however, were included. In a study of 265 adenomatous polyps Binkley et al.[3] found thirty-nine clinically benign adenomas in which foci of carcinoma were found on pathologic examination. We realize that the percentage of malignancy in our series is high, but we must consider that the average age of our patients was 64.7 years. It has been repeatedly expressed that pathologists vary a great deal in what they call early changes or atypical changes[31] and this must always be considered in any set of statistics.

It is not our intent in this study to discuss treatment of adenomatous polyps of the rectum, but we should mention briefly what treatment these 500 patients received. In many cases polyps were noted on routine examination in patients suffering from other diseases that were more important at the time than the polyps. Some of the patients refused to give consent for the polyp removal, others were dismissed from the hospital by other services before the polyps were treated, and several of the patients died from other causes before the polyps could be removed. As the result of such circumstances no treatment relative to the polyps was offered to thirty-four of the patients, while in another thirty-four only biopsy was done. Of the remainder, in

138 cases the polyps were fulgurated, in 178 cases biopsy of the polyp (which in many cases constituted complete removal) and fulguration of the base was done, while in 112 cases the polyp was removed with the electric snare and the base fulgurated. One colotomy, one anterior resection and two Miles' abdominal-perineal resections were performed.

TABLE VII

	1 yr.	2 yr.	3 yr.	4 yr.	5 yr.	Total	%
No recurrence	87	17	6	12	3	125	55.3
Recurrence (benign)	6	2	0	2	0	10	4.4
Recurrence (malignancy)	1	0	0	2	0	3	1.3
Died	..	..	..	..	..	88	38.9

As is true in most institutions, follow-up studies on these patients is most difficult to obtain and often inadequate. All patients are instructed to report to the outpatient clinic at the dispensary and are given an appointment, but many fail to keep the appointment. One worker is assigned by the social service department to the proctology clinic. She obtains daily reports from the pathology department and follows up by letter and, if necessary, by visit all patients in whom a diagnosis of adenocarcinoma or atypical changes is made. Even when contacted many of these patients fail to return for follow-up studies. Of the 500 patients in this study, it was possible to follow up only 226 patients, and of those the follow-up often was inadequate. Eighty-eight of the patients had died from other causes at the time this study was made. It is interesting to note that thirteen of the eighty-eight died of carcinoma of the stomach. We believe that this relation should be investigated.

The results of our follow-up can best be shown by a table. Table VII is the follow-up we were able to obtain on the entire group of 500 patients. The follow-up on the group showing frank carcinoma or malignant changes is more complete, more accurate and of much more importance. This group will be discussed later.

Special items on smaller groups were investigated. Of the 377 lesions submitted for histologic study, nineteen were reported as papillary (villous) adenomas, an incidence of 5 per cent. Swinton and Doane[25] found twenty papillary adenomas in reviewing 300 patients with benign polyps of the colon and rectum, an incidence of 6.6 per cent. In our review fifteen of the patients were males and only four were females. The average age in our group was 70.4 years. The level of the nineteen papillary adenomas was as follows: 1 inch level, one; 2 inch level, five; 3-inch level, seven; 4-inch level, one; 5-inch level, two; 6-inch level, two; and 7-inch level, one. No papillary adenomas were found above the 7-inch level.

TABLE VIII
MALIGNANT GROUP

Size of polyps..	1 to 3 mm.	3 to 5 mm.	5 mm. to 1 cm.	1 to 2 cm.	2 to 3 cm.	Over 3 cm.
Number	5	43	25	31	15	2
Percentage	4.1	35.5	20.6	25.6	12.3	1.6

TABLE IX
MALIGNANT GROUP

Level in inches	1	2	3	4	5	6	7	8	9	10
Number of polyps..	3	10	15	17	31	19	16	9	1	0
Percentage	2.4	8.2	12.3	14.	25.6	15.7	13.2	7.4	0.8	0

To complete our study we took as a group those cases in which the histologic report was either adenocarcinoma or atypical changes or early malignant changes, and investigated the various items we had used on the entire group of 500 patients with polyps.

The average age of this malignant group was 66.9 years as compared to 64.7 years in the entire group of patients. There were eighty-seven males and thirty females, or roughly, three to one males over females. In the total group the males were two to one over the females. These malignant polyps were equally divided as to sessile and pedunculated, fifty-eight being sessile and fifty-nine pedunculated. There were only 135 pedunculated polyps in the entire group, so then 43 per cent of the pedunculated polyps observed were malignant while only 10 per cent of the 569 sessile polyps were malignant.

A comparison of the size spread can be made by looking at Tables III and VIII. The largest percentage (35.5 per cent) of the malignant group were 3 to 5 mm. in size. From looking at these tables we can, with question, conclude that the larger the polyp the more likely it is to have undergone malignant change.

Tables IV and IX show the level at which the polyps were found in the two groups. We see

that the distribution is essentially the same, with the 5 inch level being the peak level in both groups.

The location of the polyps was essentially the same in the two groups, with equal division between anterior and posterior. There was no indication that the location of the polyp enhances its probability of being malignant. (Tables v and x.)

TABLE X
MALIGNANT GROUP

Location	Number	Percentage of Total
Anterior	26	22.4
Posterior	29	25.0
Left lateral	9	8.6
Right lateral	8	6.8
Right anterior	11	9.4
Right posterior	16	13.7
Left anterior	12	10.3
Left posterior	5	4.3

TABLE XI
MALIGNANT GROUP

	1 yr.	2 yr.	3 yr.	4 yr.	5 yr.	Total	%
No recurrence	19	6	5	7	3	40	34.1
Recurrence	4	2	..	..	..	6	5.1
No follow-up	..	..	..	..	..	43	36.7
Died	..	..	..	..	..	28	23.9

The results of the follow-up on the 117 patients with polyps that by histologic study were considered malignant are shown in Table XI. Swinton,[27] McLanahan et al.,[17] Castro et al.,[4] Binkley et al.[3] and Turnbull and Fisher[29] in 1952 have reported groups of cases followed up to ten years that have had conservative treatment of malignant rectal polyps with gratifying results. Realizing that the decision to treat these malignant polyps by conservative means is made with difficulty after much deliberation and mindful of the danger, we believe that often it is the treatment of choice for those lesions. Our results to date justify this belief, but we must emphasize that patients who have had polyps removed from the rectum by excision and fulguration are the responsibility of the operating proctologist and must be followed at regular intervals the rest of their lives.

We have presented statistics from our experience with 500 patients found to have adenomatous polyps of the rectum. Although much has been written about polyps of the large bowel that has helped in our understanding of these lesions, much more remains to be reported before the final summarizing chapter on this subject can be started.

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Neoplasms of the Small Intestine

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A RECENT case of intestinal obstruction due to intussusception, secondary to hemangioma of the jejunum, has led us to review the literature and the clinical and pathologic records of patients with tumors of the small intestine at the Boston City Hospital from 1933 through 1952.

This study has been limited to those patients whose diagnoses were found in the index file at the Mallory Institute of Pathology. The hospital record room has no index file of diagnoses during most of these years, so that patients seen on the ward and diagnosed either at operation or otherwise are not included unless a specimen was examined.

In spite of the generally accepted belief that neoplasms of the small intestine are quite rare, there have been eighty-nine microscopically proved cases in the twenty-year period previously mentioned. Sixty-two of these were autopsy specimens, the vast majority of these patients did not manifest the nature of their disease clinically, but died of incidental causes. Twenty-seven patients had enough symptoms to be subjected to celiotomy and primary resection.

During the past few years the special interest of Schatzki,[12] Golden[21] and others has made us "small bowel conscious." Because of the better understanding of the clinical manifestations and x-ray findings the diagnosis of such tumors should be considered whenever puzzling gastrointestinal pain, bleeding or obstruction occurs. Such lesions are frequently curable when the diagnosis is made and the operation performed before the obstruction or bleeding has become terminal.

Incidence. The first article on carcinoma of the jejunum was written in 1824 by Sorlin.[1] The first autopsy series of malignant lesions of the small intestine was reported by Leichtenstern[2] in 1876. They made up approximately 0.095 per cent of the malignant lesions of the entire gastrointestinal tract. Maydl[3] reported 0.029 per cent for this autopsy ratio while Nothnagel[4] found an incidence of 0.051 per cent. In 1930 Rankin and Mayo[5] reported that 0.62 per cent of all gastrointestinal malignancies were in the small intestine. Likely[6] reported an autopsy incidence of 2.3 per cent. In 1932 Raiford[7] reported 4.9 per cent for this ratio and Forgue and Chauvin[8] reported 6 per cent. Up to 1943 less than 300 surgical and autopsy specimens of carcinoma of the small bowel had been reported. Raiford[7] in 1932 reported eighty-eight cases of tumors of the small intestine among 11,500 general autopsy records and 45,000 surgical specimens from the Johns Hopkins Hospital. In 1940 Buckstein[9] found sixty-nine cases in 22,810 autopsies at Bellevue Hospital. Shallow, Eger and Carthy,[10] reviewing the literature in 1945, collected a total of 137,174 general autopsies. Among these there were 5,034 carcinomas of the large bowel, an incidence of 3.6 per cent, and 134 carcinomas of the small bowel or 0.098 per cent, but 2.4 per cent of carcinoma of the intestine. From this we can conclude that for every case of small bowel carcinoma there are approximately forty cases involving the large bowel.

Age and Sex Incidence. The youngest reported case is that of a three and a half year old child. Generally the males are involved two and a half times as often as the females and the average age is approximately fifty years.

Signs and Symptoms. There is no characteristic symptom complex which points definitely or with any degree of certainty to either benign or malignant tumors of the small intestine. Generally the symptoms are those of either obstruction or bleeding into the alimentary tract. Often the clinical picture is that of secondary anemia but some are diagnosed as cases of pernicious anemia and treated unsuccessfully as such. The symptom complex has several features in common with that of carcinoma of the large intestine, yet there are certain differences. These are more or less inde-

pendent of the site of the lesion and may be either acute or chronic in onset and may be present for variable periods of time. One of the most characteristic clinical features is that of intermittent obstruction. With high tumors vomiting occurs quite early. The vomitus usually contains blood, free hydrochloric acid and gastric enzymes. Massive hemorrhage is not frequent although the stool usually contains occult blood. The more proximal the tumor, the earlier the gastrointestinal symptoms occur. Changes in bowel habits are common, occurring usually as increasing constipation though not infrequently as alternating bouts of constipation and diarrhea. Often these take the form of recurring attacks of intestinal obstruction with severe abdominal cramps in the periumbilical and suprapubic regions, accompanied by nausea and vomiting. Weeks or months may pass between each of these attacks with periods of quiescence. Pain is common and is usually cramp-like during these periods of obstruction. Loss of weight is another common feature. Pridgen et al.[24] from the Mayo Clinic reported that 70 per cent of patients with jejunal lesions demonstrated some degree of either partial or complete obstruction of the small bowel. They report a palpable mass in 41 per cent of all cases in the jejunum and forty-eight per cent of cases in the ileum. The location of the mass in the jejunum was usually in the left lower quadrant while those in the ileum were predominantly in the right lower quadrant. Visible peristalsis was present in approximately 30 per cent of these obstructive lesions; borborygmus was present in approximately 50 per cent. About 25 per cent of the patients with small bowel tumors complained of passing excessive flatus or belching gas.

X-Ray Findings. In order to establish the diagnosis of small intestine tumors a special small intestine x-ray examination is necessary; but, of course, no barium should be given by mouth if complete or partial obstruction is present. As a prerequisite, scout films with the patient in both the upright and the recumbent position are important. Every patient with a clinical picture of gastrointestinal obstruction or bleeding which cannot be explained on the basis of a pathologic condition found in either the esophagus, stomach or the intestine should have a small intestine roentgenologic examination. In the hands of a competent roentgenologist the accuracy of both localization and differentiation is now extremely high. Weber and Kirklin[11] reported correct diagnoses in 94 per cent of duodenal neoplasms, 85 per cent of jejunal neoplasms and 67 per cent in the ileum in a series of sixty-two small intestine tumors.

Generally speaking three separate and distinct methods are available to the roentgenologist in the study of these tumors. The first is the small intestine enema as described by Schatzki.[12] This consists of passing a Levin tube into the second portion of the duodenum and filling the small intestine with a barium suspension which is allowed to run through slowly.

The second method is a modified small intestine enema. A Miller-Abbott tube is passed into the small intestine as a therapeutic measure and the patient is decompressed. A small amount of thin or diluted barium suspension is then introduced just proximal to the obstruction and fluoroscopically examined.

The third method is called a motility series. Thirty grams of barium sulfate are given orally at 8 A.M. and films are taken one-half hour later and repeated at half-hour intervals. These serial films are examined immediately and the patient is fluoroscoped and spot films are taken as indicated. When all of the small intestine has been well visualized and the head of the column has reached the cecum, the examination is terminated.

Benign lesions of the small intestine are difficult to diagnose before any obstruction has occurred clinically. Large benign lesions involving one portion of the thin-walled structure may be hidden by the flexibility and normal appearance of the uninvolved circumference. With a non-obstructive intraluminal lesion, subtraction of the normal lumen may be expected. In the duodenum this is easy to find but in the jejunum and ileum it must be actively sought. Extraluminal growths are also difficult to diagnose early and usually can be diagnosed roentgenologically only after they have reached sufficient size to cause obstruction by pressing against the wall of the intestine, manifesting a large intraluminal radiolucent defect. In the presence of malignant tumors various degrees of filling defects are demonstrated. Usually, this is of an annular constriction.

Pathology. There is some difference of opinion as to the relative frequency of the two principal groups of malignant tumors of

the small intestine, carcinoma and sarcoma. Fraser,[13] Mayo,[19] Cameron[14] and others report that carcinoma was only slightly more frequent. This is a very different situation from that of the large intestine or the stomach where carcinomas make up over 95 per cent of the cases. Lymphosarcoma has been the most frequently reported type of sarcoma involving the small intestine.

Carcinoma. The involved portion of small bowel is usually quite short, rigid and often fixed. Proximal to this lesion the intestine is frequently distended. At celiotomy the carcinoma is usually found to have encircled the bowel completely in an annular or napkin-ring type of tumor and often involves the small intestine for a relatively short distance of 1 or 2 inches; frequently this involvement may be simply a fraction of that area. By the time the tumor has been explored at celiotomy it has usually brought about complete or nearly complete occlusion of the small bowel with subsequent intestinal obstruction. The late appearance of these clinical features is accounted for by the fluid nature of the bowel contents in the small intestine and, therefore, many of these patients first present themselves as cases of chronic or acute intestinal obstruction of unknown etiology. As a consequence of this gradual obstruction, hypertrophy and dilation of the proximal loop is a frequent finding at celiotomy. Occasionally the carcinoma presents itself clinically as an intussusception of the small bowel which at operation is not completely reducible. However, intussusception is more frequently an occurrence in polypoidal types of tumors.

In spite of the fact that the intestinal symptoms develop relatively late, secondary metastasis occurs quite early. This is probably due to the abundance of the lymphatic drainage and the vascular supply along with the absorptive functions of the small intestine. Metastases occur to the related lymph nodes and peritoneum, the liver, lungs, long bones and to the dura in that order of frequency. In 1953 Ebert et al.[23] reported from the Boston City Hospital on tumors of the duodenum with detailed case reports of two recent operations for carcinoma of the duodenum. Some of their cases are also included in this report.

Treatment. The treatment of choice for any tumor or type of malignancy in the small intestine is resection of the afflicted loop of bowel along with a wide wedge of related mesentery and contained lymphatics. The continuity of the small intestine is re-established preferably by end-to-end anastomosis. Because of its anatomic relationship the duodenum, however, presents a much more complicated surgical problem than neoplasms of the ileum or jejunum. If the neoplasm manifests itself as acute intestinal obstruction, it is essential that these patients be decompressed to a maximum degree before operation. Their fluid and electrolyte balance, anemia and vitamin and plasma protein deficiencies should be improved as much as possible preoperatively.

Prognosis. Prognosis is good in cases of carcinoids and poor in cases of carcinoma and sarcoma. In 1947 Botsford and Seibel[18] reported an average survival of eleven and a half months among eighteen cases of carcinoma and six months among thirteen cases of sarcoma. In Cameron's[14] series in 1937 the average duration of life was 17.6 months following the resection. In Mayo and Nettrour's[19] thirty-one cases of jejunal carcinoma reported in 1937 two patients survived for a period of seven years. In 1932 Wakely[20] reported a patient living and well eight years after resection of the terminal ileum for adenocarcinoma. This was unexpectedly found while exploring the abdomen for an umbilical hernia.

The results of surgery for small intestinal malignancy compare quite favorably with those of the large bowel. Since the malignancies of the small intestine metastasize quite early and since the diagnosis is delayed because of the relative absence of symptomatology until late in the stage of the disease, it is usually beyond the point of operability when explored. Carcinoma of the small intestine seems to have a better prognosis than that of sarcoma. Because of the anatomic location and relationship of the duodenum, primary malignancy in the duodenum is less favorable than that of the jejunum or ileum.

PATHOLOGIC MATERIAL STUDIED

After reviewing the clinical and pathologic records of the proved neoplasms of the small intestine it was decided to present the data concerning the cases found incidentally at autopsy separately from the rest of the cases. There were sixty-two in the group of those found at autopsy. Table 1 shows the locations in the intestine of the autopsy patients and shows

the different kinds of malignant tumors found in these locations. Table II presents the same type of data for the benign lesions found at autopsy. It is interesting to note that practically three-fourths of these lesions found at autopsy were in the benign group, indicating that when a patient has a symptomless tumor in the intestine it is most likely to be benign. The data found by further study of this rather miscellaneous group of patients were found to be relatively insignificant.

TABLE I
LOCATION OF MALIGNANT TUMORS IN AUTOPSY CASES

	Duodenum	Jejunum	Ileum	Total
Adenocarcinoma	8	2	1	11
Leiomyosarcoma	1	0	1	2
Lymphosarcoma	0	0	1	1
Malignant adenoma	0	0	0	0
Melanosarcoma	0	0	0	0
Reticulum cell sarcoma	0	0	2	2
Total	9	2	5	16

TABLE II
LOCATION OF BENIGN TUMORS IN AUTOPSY CASES

	Duodenum	Jejunum	Ileum	Total
Adenoma	0	2	0	2
Fibroma	0	2	0	2
Ganglioneuroma	0	2	1	3
Hemangioma	2	3	2	7
Leiomyoma	4	6	10	20
Lipoma	4	4	3	11
Lymphangioma	0	0	0	0
Neurofibroma	1	0	0	1
Polyp	0	0	0	0
Total	11	19	16	46

TABLE III
AGE AND SEX DISTRIBUTION

Females (14)	1	0	1	2	2	3	4	1
Males (13)	1	1	4	0	0	2	5	0
Age	0–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80

Operative Cases. There were twenty-seven patients in this group. Table III shows their age and sex distribution. In this table the malignant and non-malignant are both included and are not shown separately because the numbers are too small. It is of great importance, however, to notice that ten patients, or more than one-third, are under the age of forty.

Table IV presents the data as to the location of the tumor and the type of malignancy in the operative cases. There were sixteen patients in the group, of whom more than half had adenocarcinoma. Exactly half of all the patients had the lesion located in the jejunum.

TABLE IV
LOCATION OF MALIGNANT TUMORS IN OPERATIVE CASES

	Duodenum	Jejunum	Ileum	Total
Adenocarcinoma	3	5	1	9
Leiomyosarcoma	0	3	0	3
Lymphosarcoma	0	0	0	0
Malignant adenoma	0	0	1	1
Melanosarcoma	0	0	1	1
Reticulum cell sarcoma	0	0	2	2
Total	3	8	5	16

TABLE V
LOCATION OF BENIGN TUMORS IN OPERATIVE CASES

	Duodenum	Jejunum	Ileum	Total
Adenoma	0	2	1	3
Fibroma	0	0	1	1
Ganglioneuroma	0	0	0	0
Hemangioma	0	3	0	3
Leiomyoma	0	0	0	0
Lipoma	0	1	0	1
Lymphangioma	0	0	1	1
Neurofibroma	0	0	0	0
Polyp	0	0	2	2
Total	0	6	5	11

Table V presents similar data concerning benign tumors in operative cases. Here there was no preponderant type but it is interesting that no lesions were found in the duodenum of eleven in this group.

It can be seen that in the patients with lesions found at operation 69 per cent had malignant diseases compared to 26 per cent of malignant disease found at autopsy.

Unfortunately, the clinical records of six cases in 1933 were unsatisfactory for further

study. Therefore the data on symptoms were tabulated from the remaining twenty-one.

Symptoms. As seen in Figure 1, only eight of twenty-one patients complained of preoperative constipation in spite of the fact that there was intestinal obstruction that was incomplete in eleven patients and complete in five additional cases. Nineteen of twenty-one patients complained of acute pain, eighteen of nausea and sixteen of vomiting. There was a definitely related loss of weight in six cases. We believe that an outstanding clinical manifestation is the mass palpable in eleven, or over 50 per cent of the cases. In 33 per cent there was visible peristalsis. Again over 50 per cent had definite melena although the red blood cells and hematocrit were not significantly depressed except in three cases.

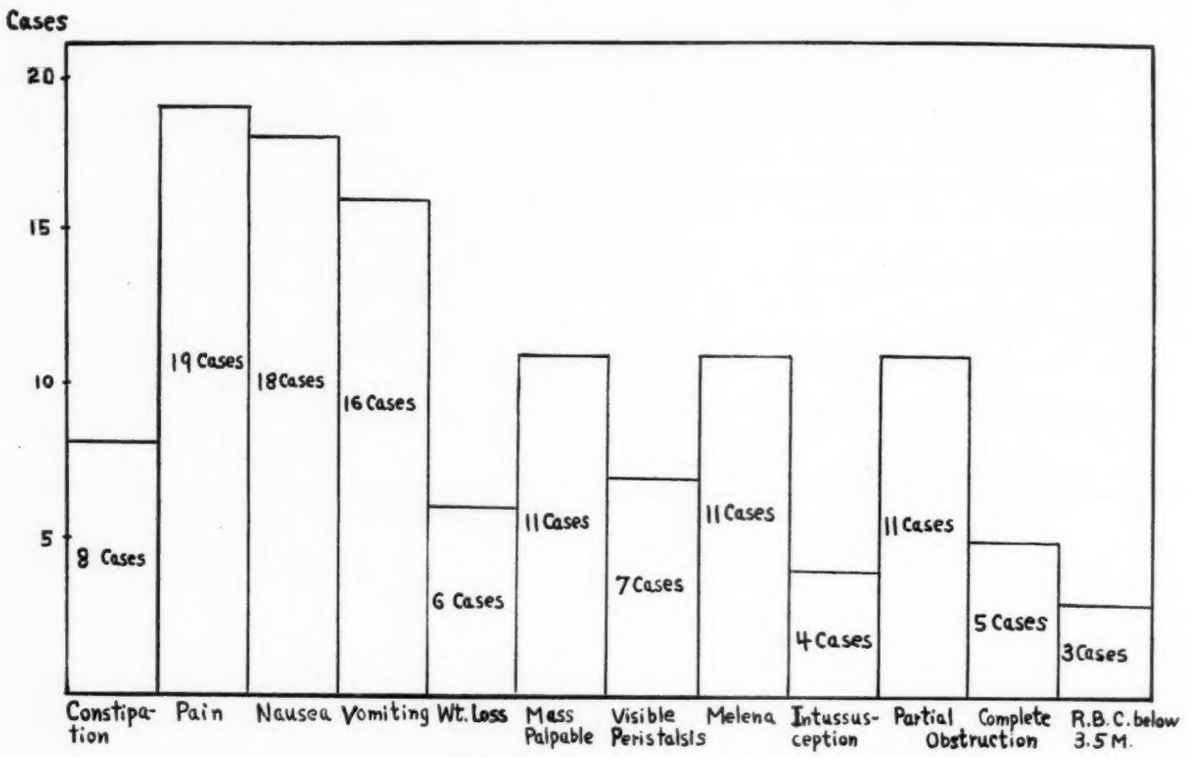

FIG. 1. Symptomatology of twenty-one cases with adequate records.

Because our last clinical case presented as an intussusception, we were especially on the lookout for this in the previous cases but could find a total of only four cases or less than 20 per cent.

Treatment. Twenty-one operations were performed, of which twenty consisted of resection with primary anastomosis and one was a biopsy for a patient with hemangioma of the small intestine.

Three of the patients died postoperatively. One had malignant adenoma of the ileum and died of a subdiaphragmatic abscess. The second postoperative death was a patient with adenocarcinoma of the duodenum which was resected. A jejunal fistula and peritonitis developed. Both of these deaths occurred in 1938, in the pre-antibiotic era.

The third death, which occurred in 1947, was that of a patient who had reticulum cell sarcoma of the ileum and who died after an extreme reaction secondary to intravenous protein hydrolysate feeding.

The remaining eighteen patients recovered without major complication and left the hospital in good condition.

SUMMARY

Delay in the diagnosis and treatment of small bowel tumors occurs quite frequently and is due to the fact that the clinical manifestations occur late in the disease because of the liquid character of the bowel contents in

the small bowel. Often the patient is presented as a case of intestinal obstruction or intussusception, the obstruction often being of an intermittent type. In the presence of anemia of gastrointestinal origin in which the stomach and large bowel have been thoroughly investigated, a radiologic examination of the small bowel is indicated.

Twenty-seven cases have been recorded, with three deaths. In spite of the fact that many patients will probably continue to come to surgery late in the course of the disease, a very small immediate mortality should be encountered in future operations of this nature.

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Wounds of the Diaphragm, Stomach and Duodenum

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The diaphragm, stomach and duodenum are located in the mid-portion of the torso, at the approximate center of gravity. The steering device of modern vehicles is centered about these structures. It is remarkable that these organs are injured so infrequently.

One would expect both direct and indirect trauma to exert their maximum damage in this region. Fortunately, this is not the case. Nevertheless, with trauma to any of these organs, a serious chain of events follow. The diagnosis must be established and treatment instituted promptly.

DIAPHRAGM

Wounds of the diaphragm may be produced by direct or indirect trauma, or by inflammatory necrosis of the diaphragm.[1] Indirect trauma may produce a hernia in any portion of the diaphragm. Crushing is the usual force producing such an injury. These accidents occur in landslides, mine disasters, automobile and airplane crashes, and falls from ladders.

Veterinarians are unusually alert to traumatic injury to the diaphragm. A dog, struck by an automobile, that sits on its haunches and pants for breath is suspected of having a traumatic diaphragmatic hernia. A chest plate and a barium swallow are almost routine procedures following such an accident. Surgeons can well afford to emulate the veterinarians. Diaphragmatic hernia should be considered in patients having indirect trauma to the abdomen.

Direct trauma may produce a hernia in any portion of the diaphragm. A penetrating wound, such as that produced by a gun or knife, is the most common form of trauma. A small perforation may be made in the diaphragm and the clinical findings will be minimal or absent. At a later date, with a sudden increase in intra-abdominal pressure, herniation of abdominal viscera may occur through the small defect in the diaphragm which had been asymptomatic. This sequence of events may be expected in soldiers who received penetrating chest wounds. Hughes et al.[2] report a soldier who received a penetrating chest wound and thirteen months later, while carrying heavy sacks, he was seized with severe upper abdominal pain. At operation, he had almost complete herniation of the stomach into his chest.

Inflammatory necrosis of the diaphragm may develop from a subdiaphragmatic abscess. Perforation of the diaphragm may follow necrosis produced by a drainage tube which has been placed in empyema cavities and left there for long periods of time.

Clinical Findings. The clinical findings in wounds of the diaphragm will vary with: (1) the amount of impairment of normal function of the diaphragm; (2) the amount of increased intrathoracic pressure, which produces impairment of circulation and respiration; (3) the structures involved in the hernia; and (4) the degree of mechanical interference produced.

Treatment. Wounds of the diaphragm require immediate surgery, if there is any indication of an associated injury to a hollow viscus. This situation holds particularly in those patients having direct trauma.

Following indirect trauma surgery may be delayed, if there is no evidence of injury to other structures, until the acute changes caused by the injury have been corrected.

The principle involved in the repair of wounds of the diaphragm is to restore physiologic function and to return the organs to their normal habitat.

The transthoracic or abdominal approach, or a combination of the two, may be selected for the repair,[1,3] The technic chosen depends

upon the size of the diaphragmatic defect and the type, situation and amount of abdominal viscera involved in the hernia.

Paralysis of the diaphragm is advisable in the repair of large defects. This is accomplished by temporary crushing or permanent interruption of the phrenic nerve.

In large defects of the diaphragm, or with loss of tissue, thoracoplasty may be indicated. The diameter of the chest is narrowed sufficiently to permit closure of the defect.

STOMACH

All wounds of the stomach will play "second fiddle" to that received by Alexis St. Martin on June 6, 1822, at Fort Mackinac, Michigan. Beaumont's observation and study of this gastric fistula forms the basis of modern gastric physiology. St. Martin displayed his fistula in various parts of the country and had offers to exhibit it in London and Paris. He fathered seventeen children and died in 1880, at seventy-six years of age.

Sir William Osler attempted unsuccessfully to secure the stomach for the Army Medical Museum. The St. Martin family allowed the body to putrefy by exposure and buried it 8 feet underground in the Canadian Woods.[4]

The stomach is an organ of varying size, distensible and somewhat mobile. Traumatic rupture of the stomach is rare, without an associated penetrating wound.

It is an interesting clinical axiom that alkalies are more damaging to the esophagus and acids more destructive to the stomach. Acids in high concentration, however, can be injurious to the esophagus as well as to the stomach.

In a patient who has swallowed a corrosive, the use of a stomach tube carries some risk, due to the softening of the tissue and the danger of perforation. Emetics are dangerous for the same reason. Neutralization with an alkali should be considered. Alkaline carbonates, which liberate carbon dioxide, should be avoided because of the gaseous distention of the injured stomach. With associated edema of the glottis, tracheotomy may be necessary.[5]

Surgical treatment is indicated, if sequelae follow the ingestion of acid. Resection is the procedure of choice. Other technics, such as pyloroplasty, dilatation of the pylorus and gastro-enterostomy are discouraged, because of the difficulty encountered in suturing the damaged layers of the stomach wall.[6,7]

Treatment. Laparotomy is indicated whenever a penetrating wound of the stomach is suspected. It should be done as quickly as the patient can be prepared for surgery. A Levin tube is always inserted before operation. If the aspirate contains fresh blood, a stomach wound may be suspected. An upright x-ray examination may reveal free air beneath the diaphragm.

Perforation of the posterior wall and the fundus of the stomach are frequently overlooked. The posterior wall can be seen and examined by dividing the gastrocolic omentum and elevating the decompressed stomach. A distended stomach is difficult to examine. Continuous decompression is necessary throughout the operation.

The irregular, devitalized edges should be excised. The wound is converted to a linear incision and closed.

DUODENUM

The location, size and course of the duodenum protect this organ from a great deal of trauma. Nevertheless, retroperitoneal tear of the duodenum is overlooked more frequently than trauma to any other portion of the gastrointestinal tract.

Rupture of the duodenum accounts for 10 per cent of all gastrointestinal ruptures. Approximately one-third of the tears are in the retroperitoneal portion.[8–10]

Indirect trauma, such as a kick by a horse or a blow while boxing, is the usual force which produces the lesion. The tear develops from a sudden increase in the intra-abdominal pressure. The retroperitoneal portion of the duodenum is fixed and immobile.

The signs of retroperitoneal tear are obscure and may be delayed in their appearance. X-ray examination may reveal evidence of retroperitoneal emphysema. Pain in the testicles is characteristic of retroperitoneal duodenal trauma.[11] The testicular pain is due to bile irritation of the sympathetic nerve chain which accompanies the spermatic artery. Pain in the posterior part of the neck, when pressure is applied to the abdomen, is another helpful sign of retroperitoneal tissue damage. A patient with persistant hypogastric pain and increase in pulse rate, following a blow in the epigastrium, should be

suspected of suffering from a rupture of the duodenum in the retroperitoneal portion.

At laparotomy the presence of retroperitoneal edema or bile extravasation about the duodenum indicates the necessity of exposure of the retroperitoneal portion of the duodenum.[12–15]

Clinical Findings. (1) Pain is usually present in the epigastrium. (2) Rigidity may or may not be present. (3) Pain is present in the testicles. (4) There is pain in the back of the head with pressure on the abdomen. (5) Air or gas is present in the retroperitoneal space. (6) Air in the region of the right kidney suggests retroperitoneal tear of the duodenum.

Surgical Treatment. The best exposure of the duodenum is obtained by wide mobilization of the hepatic flexure of the colon. The hepatic flexure is freed by dividing the suspensory bands that run from the lower portion of the right lobe of the liver and the lateral abdominal wall. The right portion of the gastrocolic omentum may need separation.

The second portion of the duodenum may be mobilized by dividing the peritoneum lateral to the duodenum. The third portion requires careful dissection to prevent injury to the superior mesenteric vessels and the mid-colic artery.

The duodenum can be elevated and rolled medially for thorough inspection.

The hole in the duodenum is closed with interrupted sutures, and a drain placed in Morrison's pouch. A catheter jejunostomy will permit feeding so that the duodenum may be kept at rest for several days. The stomach is decompressed with a Levin tube for several days.

SUMMARY AND CONCLUSIONS

1. Wounds of the diaphragm are produced by direct or indirect trauma and inflammatory necrosis of the diaphragm.

2. The principle involved, in the repair of wounds of the diaphragm, is to restore physiologic function and to return the organs to their normal habitat.

3. St. Martin's wound of the stomach is the most famous of all gastric wounds.

4. Wounds of the posterior wall and fundus of the stomach are overlooked frequently.

5. Retroperitoneal tear of the duodenum by indirect trauma is overlooked more frequently than trauma to any other portion of the gastrointestinal tract.

6. Pain in the testicles is characteristic of retroperitoneal duodenal tear.

7. The principles of exposure of the duodenum are enumerated.

8. Retroduodenal tears must be repaired and the area placed at rest until healing occurs.

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Pleurodynia (Coxsackie Virus Infection Group B) and Acute Abdominal Condition in Children

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THE occurrence of acute abdominal symptoms during the prodromal period of the usual childhood diseases is common. The differentiation from acute appendicitis is at times impossible in the absence of skin lesions or other signs and symptoms suggesting the correct diagnosis. Not infrequently an acutely inflamed appendix is found which is coincidental. I have reported a case of gangrenous appendicitis in a patient with mumps, and I have also had one patient with proven appendicitis who had poliomyelitis and subsequently developed paralyses. Cases of acute appendicitis are not infrequently seen following epidemics of "flu" and "grippe." These, however, are not common and many patients are operated upon in whom little is found except occasionally some enlarged mesenteric lymph glands. In known epidemics the diagnosis is considered but in other cases the operation is performed needlessly. The finding by Dalldorf[3] in 1947 of the Coxsackie viruses has brought a new clinical entity to common knowledge which must be considered in the differential diagnosis. Experience with four cases in the course of a few months would indicate that this entity is extremely common and should be known to all surgeons as well as to pediatricians and general practitioners.

While investigating epidemics of poliomyelitis, Dalldorf[3] isolated twenty-eight different strains of a virus from the stool and pharyngeal washings of two boys in Coxsackie, New York. Twenty of these were in a group which he called A; the others were called group B. They resembled the viruses of poliomyelitis and murine encephalomyelitis, and were resistant to ether, penicillin, streptomycin and chloramphenicol. He identified these by injecting the prepared solution into suckling mice. In group A they became weak and hopelessly paralyzed and died with severe destructive lesions in the muscles. In group B, the mice became spastic, tremulous or paralyzed. The brain tissue was softened and the fat pads conspicuously pale. Group A contained serologically distinct types but the B strains were related. To date fifteen of the strains demonstrated have been found to be clinically important. In some of the proven cases neutralizing antibodies were found in the serum following the infection. The exact time this immunity lasts is not known, but in two cases[10] this disappeared in eight months. These antibodies have not been found in all cases of proven C virus infection. For instance, Disney[4] found only nine positive cases in seventeen tested. These viruses occur in multiple types and are possibly the most common virus of man with the exception of the influenza group. A and B types may be found in the same patient and at times neither may be found when the clinical diagnosis is sure. They may be present in patients without symptoms. Epidemics due to these viruses have been reported widely throughout the world. The reported epidemics are mostly in August, September and October, but some have been reported in June and sporadic cases may occur at any time.[8] Attacks commonly occur in children but are present in adults. Attacks are usually self-limited and are not fatal. It is believed that infection is spread by contact only and that repeated attacks occur during the average lifetime. The use of aureomycin has been reported as being of value.[8]

Group A causes the disease known as herpangina which is characterized by fever, headache, etc., and is diagnosed by minute ulcerative lesions which occur on the posterior part of the mouth and throat. Group B causes epi-

demic pleurodynia, and this disease is important to the surgeon. It was first observed by Finsen in 1863 and again in 1865, and was first described by him in 1874.[5] Other epidemics have been reported as "epidemic resembling dengue fever," "aseptic meningitis," "epidemic myalgia," "summer grippe," "intercostal neuritis," etc. The attack is ushered in after a prodromal period of two or three days of malaise, anorexia and vague general symptoms, by severe pains with prostration which may be mild to severe, fever, headache, cough, muscle aches and pains, and pleuritic signs. Any of these symptoms may be present or absent. The attacks usually last from three to five days, to two to three weeks. Severe weakness follows the attack out of proportion to the severity and a slow convalescence is common. This last statement is not true of the abdominal attacks seen in children.[7] As reported in the literature these symptoms may become chronic and severe, and it is in this type that muscle biopsy may give the diagnosis.[1] No fatalities have been reported.

TABLE 1
CLINICAL FINDINGS IN TYPICAL CASES

	Medical		Surgical		Total	
	No. of Cases	Per cent	No. of Cases	Per cent	No. of Cases	Per cent
	55	100	49	100	104	100
History of contact	16	29	9	18	25	24
Average age	6½ yr.		4¾ yr.		5½ yr.	
Onset acute	38	69	46	94	84	82
Site of pain:						
Chest	9	16	0	0	9	9
Abdomen	36	65	48	98	84	81
Both	10	18	1	2	11	11
Red throat	33	60	14	29	47	45
Follicles on tonsils	3	5	1	2	4	4
Cervical glands enlarged	18	33	22	44	40	38
Respiratory system:						
Dyspnea (including grunting)	30	55	8	16	38	36
Cough (including grunting)	3	5	5	10	8	8
Alae nasi active (including grunting)	9	16	6	12	15	14
Pleural rub	7	13	0	0	7	7
Crepitations	7	13	3	6	10	10
Abdominal signs:						
Generalized tenderness	20	36	15	30	35	34
R.I.F. tenderness	3	5	10	20	13	13
Subjected to laparotomy	0	0	9	18	9	9
Central nervous system:*						
Headache	15	27	1	2	16	15
Drowsiness	4	7	0	0	4	4
Meningism	6	11	0	0	6	6
Average duration of acute phase	4½ days		5½ days		5 days	
Recrudescence	27	49	21	43	48	46
White count less than 10,500 per cu. mm	35/38	92	12/14	86	47/52	90

* Seven cases (including the six with meningism) had cerebrospinal fluid examination and in only one was it abnormal, with 40 cells per cu. mm. (Disney et al.[4])

Clinical reports show a varying but definite number of cases in each outbreak of pleurodynia which present symptoms suggesting an acute condition of the abdomen. Table 1 gives a breakdown of an epidemic in England. One hundred four patients were studied and of

these eighty-four (81 per cent) had some abdominal pain. Forty-nine were seen by a surgeon. Thirty-five (34 per cent) complained of generalized abdominal tenderness with thirteen (13 per cent) localized in the right lower quadrant, a total of forty-nine (47 per cent). Nine (9 per cent) were subjected to laparotomy, with essentially normal findings. Of the 114 patients seen at Boston City Hospital[5] in 1947, tenderness was confined to the abdomen in thirty-three. Nine patients were admitted to the surgical ward but only one was operated upon with negative findings. Warin et al.[12] reported 138 (52 per cent) of 262 cases which showed abdominal pain to be the main presenting symptom. The pain may be in the epigastrium or in either upper quadrant as well as in the right lower quadrant and the umbilical region. No cases of pain in the left lower quadrant were reported. In ten of Disney's[4] cases, the pain began at the umbilicus and then moved to the right lower quadrant. Seventeen patients had initial pain in this area. The diagnostic problem is self-evident.

The type of pain is typical in that it is in the muscle rather than deep and comes on in spasms with relatively normal periods between. The usual story is an exacerbation of pain lasting from five to ten minutes followed by about half an hour of relative relief. Another characteristic of the pain is that it is aggravated by any movement, coughing, sneezing or even talking. Nichamin[8] emphasizes that a younger child has more difficulty in describing pain and discomfort. Children under five seldom complain of headache but parents will pick up any abdominal distress and notice any changes in bowel habits or lack of appetite, etc. Frequently there will be a history of a varying degree of abdominal pain for several days, finally becoming so severe that the child is brought to the doctor. In the presence of an epidemic the diagnosis is not difficult; but when isolated cases are seen or when first seen in the hospital, there is a real clinical problem.

In the combined abdominal and thoracic type the illness may begin with abdominal pain followed by chest pain or vice versa. In this type the waving of the alae nasi as well as the grunting type of respirations on motion should cause one to suspect the diagnosis. Chest films will be negative. Other symptoms which are common in the usual childhood diseases are present, such as elevation of temperature from 101° to 103°F. (Table 1.) The temperature may be initially high and entirely out of proportion to the abdominal signs and may drop to normal in a few hours. Warin[12] states that "Characteristically, the temperature dropped to normal or near normal when the severe spasmodic pain ceased." Vomiting was usually brief and occurred most often either at the onset of the acute abdominal or chest pain or at the same time as the severe headache of a developing meningitis.[4] The blood count was characteristically normal or low with an increase in the polymorphonuclear count. Seventy-six per cent of the cases at Boston City Hospital showed a normal white count on the first day, subsequently dropping below 5,000. Forty per cent had a 3 per cent eosinophil count at some stage of the illness, usually appearing about the fourth day. The white count in the cases presented by Disney[4] is summarized in Table 1. Other symptoms noted were pains in the limbs, cramps in the feet and paresthesias of the limbs or body. Swollen glands were also quite common.[4]

CASE REPORTS

CASE 1. A ten year old boy was admitted to the hospital on July 28, 1952, complaining of abdominal pain of twenty-four hours' duration. Since that morning he had also had difficulty in breathing. The patient's mother stated that he had been complaining of abdominal pain off and on for several weeks. Temperature was 101°F., pulse 120 and respirations 28.

When seen the patient did not look acutely ill. He lay quietly in bed, breathing normally. It was noted that the alae nasi moved with respiration and that on moving or talking he had an expiratory grunt. For example, in answer to a question about pain he said, "my stomach hurts"—grunt. He answered each time in this manner so that I was sure he had a pathologic condition in his chest. X-ray of the chest, however, was negative. The urine was normal. The white blood count was 9,650 with 85 per cent polymorphonuclears. Examination showed localized muscle spasm in the right lower quadrant but no definite rebound. Rectal examination was unsatisfactory; no mass was felt. I could not decide whether the abdominal pain was coincidental to some systemic condition or due to appendicitis. Subsequently he was seen by Dr. Mary Fayala,

who made a diagnosis of pleurodynia, but who also had a spinal tap done which showed twelve lymphocytes and normal pressure. The child was put on aureomycin and his temperature dropped in twenty-four hours and fluctuated 1 degree for a couple of days. The second day he complained of pain in both flanks and neck. He held his head back and said it felt better that way. He was discharged as well on August 5, 1952.

CASE II. This six year old boy was seen at the hospital with Dr. I. Rosefsky at 3 P.M. on August 8, 1952, with a history of severe abdominal pain of eight hours' duration; his temperature was 101°F., pulse 88 and respirations 28. White blood count was 11,750 with 84 per cent polymorphonuclears. Urinalysis was normal. Examination showed marked muscle spasm with some rebound, but waving of the alae nasi and grunting expiration were noted and watchful waiting advised. There was marked injection of the throat. X-ray of the chest was diagnosed possible miliary pneumonia. At 8 P.M. the temperature was 103°F. but the abdomen was soft. The next day the temperature was normal and the patient was discharged. Subsequently, he had several abdominal attacks during the next few weeks. He has been well to date.

CASE III. A four year old white girl was seen at night with Dr. James Johnson on August 31, 1953, complaining of acute abdominal pain of six hours' duration with nausea but no vomiting. This child also showed the movement of the alae nasi and the expiratory grunt on motion, but the abdominal muscle spasm was localized in the right lower quadrant. There was no rebound tenderness. The white count was 8,300 with 74 per cent polymorphonuclears. The urine (not catheterized) showed 18 to 20 white blood cells per high power field. X-ray of the chest was negative. Despite the possibility of pleurodynia, operation was decided upon at the parents' urging and a normal appendix with mesenteric lymphadenitis was found. The pathologic report was chronic lymphadenoid appendicitis, merely indicating hyperplasia of lymphoid tissue.

CASE IV. A six year old boy was seen with Dr. Vesta Rogers at the hospital about 2 P.M. on September 7, 1953, with a history of nausea and fever of two days' duration. His father stated that two days before he had complained of tiredness and some fever, about 102°F. At 9 A.M. on this date he began vomiting and had been vomiting off and on since then. About one month before one of his brothers had contracted poliomyelitis and before admission this had been considered a possibility. X-ray of the chest was clear and a spinal tap was negative. The spinal fluid showed one lymphocyte. The white count was 6,850 with 87 per cent polymorphonuclears. When seen his temperature was 103°F., respirations 20, pulse 100. His face was quite flushed but despite the complaints and the temperature he did not look very ill. On questioning, the pain was severe about the umbilicus and to a lesser degree in the right upper and lower quadrants. There was no waving of the alae nasi and no grunting respirations. There was slight, if any, muscle rebound and it was noted that the pain was aggravated by motion or pressure and that the child seemed quite comfortable between spasms. In view of the foregoing, a tentative diagnosis of pleurodynia of the neurogenic type was made and watchful waiting advised. The following day his temperature was normal and he was generally better. He still had some abdominal distress and complained of severe muscle pains about the hips and limbs. X-rays of the femurs were negative for osteomyelitis. Convalescence was uneventful and subsequently Dr. Rogers saw several children with severe abdominal complaints in the same neighborhood, which she considered to be pleurodynia.

COMMENTS

In a résumé of my four cases it is noted that the chest symptoms were present if one looked for them. However, when the patient was first seen by the attending physician and also by myself, and when first brought to the doctor by the patient's family, the complaints were entirely abdominal. It is only on careful examination that the possibility of a Coxsackie virus infection is suspected. The findings were essentially the same, with a comparatively normal white count with a moderate polymorphonuclear increase. There was an increase in respirations with localized pain which was usually about the umbilicus or over McBurney's area, with muscle spasm but no rebound. It is very probable that a great many of the appendices which we see in some of our so-called epidemics of colds and summer "grippe," etc., are really due to, or the symptoms are caused by, a Coxsackie virus infection. The fact

that only one member in the family is affected and that it usually occurs in epidemic form does not rule out the condition. It is my impression that the alae nasi motion with the grunting type of respirations should be looked for in all children who have a fever and abdominal pain and that this virus condition should be considered. A suspected diagnosis will relieve the surgeon of a great deal of anxiety but it is very essential, as brought out by the British observers,[12] that a case of appendicitis not be missed. In retrospect, I remember one of the older surgeons telling me about an epidemic of appendicitis, so-called, in which he operated upon two members of a family before he realized that it was not appendicitis from which they were suffering.

It is also noted that these four cases occurred in July, August and September, which corresponds with the time when the virus seems to be the most active. These children were from different parts of the city and one was from out of town. The close correlation with infantile paralysis is evident as borne out in the literature and is of no importance, except that it gives a good prognosis to a patient who would otherwise be considered seriously ill. In my practice I have seen many such cases; and whether or not my decision was to remove the appendix, I thought that I had failed in some way because I did not know the underlying pathologic condition. I am sure that all surgeons have had a similar experience.

CONCLUSION

Coxsackie virus infection is a widespread systemic disease of epidemic nature occurring seasonably in epidemic form, which should be made familiar to all members of the medical profession. While seen most frequently by general practioners and pediatricians, the reports in the literature indicate that a considerable percentage of the cases have such acute abdominal signs and symptoms that they are referred to a general surgeon for differentiation from acute appendicitis. The finding of the virus in the stool and pharyngeal washings requires a laborious and time-consuming technic which is not suitable in acute cases, and a diagnosis must be made clinically. In actual practice the mere addition of this disease to the discussion of the possible diagnoses will often clarify the situation. In those cases of suspected C virus infection in which appendicitis cannot be ruled out surgery can be performed with no fear of unusual complications.

Since this condition is self-limited, if so desired in doubtful cases one can embark upon a period of watchful waiting without antibiotics and with greater tranquility without jeopardizing the patient. The injudicious use of sulfonamides and antibiotics in children with abdominal pain has in my experience resulted in the later appearance of appendiceal abscess or intestinal obstruction because of missed appendicitis with perforation.

Mesenteric lymphadenitis has been thoroughly investigated with no definite conclusions as to a method of differential diagnosis from acute appendicitis. We are at present at the Binghamton City Hospital taking biopsies of rectus muscle in all cases in which the diagnosis of acute appendicitis has been made and in which the findings are essentially normal. This is in line with the findings reported by Brown[1] of lesions typical of Coxsackie virus infection in his two reported cases. To date our findings have been negative. This is expected inasmuch as the infection is not of sufficient duration to produce pathologic changes except in the severe chronic types. It should be emphasized that the disease tends to recur and that repeated attacks with the same symptoms are common.

SUMMARY

1. A brief résumé of the history and present knowledge of Coxsackie virus infections has been presented, with special emphasis on group B infections (pleurodynia).

2. The frequency of acute abdominal signs and symptoms has been shown as noted in the literature and four clinical cases reported.

3. The history, the elevated temperature and the clinical picture indicative of pleurodynia have been presented and a means of differential diagnosis from acute appendicitis has been suggested.

4. In cases which cannot be differentiated from acute appendicitis surgery is indicated and can be done without fear of complications.

5. The medical profession, including the surgeon, should be made aware of this clinical entity in order to prevent unnecessary surgery.

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If a suspicious nodule is present in the rectum, we must not jump to the conclusion it is a malignancy. Infection with Endamoeba histolytica may occasionally produce a hyperplastic mass here (with or without abscess formation) and may even cause obstructive symptoms. The condition should be thought of because conservative treatment often suffices. (*Richard A. Leonardo, M.D.*)

Atypical Neuralgia of the Chest Simulating Angina Pectoris

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STUDIES by Rinzler and Travell[1] in 1948 on the somatic component of cardiac pain have shown that in some patients with angina pectoris and acute myocardial infarction, pain is relieved by blocking secondary trigger areas in the skeletal muscles of the chest. They believe that the myofascial component[2] may be the major factor in maintaining a continuing pain cycle. They[3] emphasize that the main source of irritation in the pain syndromes involving the chest are sprains and strains at the origins of the pectoralis major and minor muscles, and sometimes at their insertions as well.

It is true that these cases of post-traumatic pain syndromes involving the chest are unusual and few in number compared with cases of true angina pectoris, yet the fact that these pain syndromes do occur makes their recognition important, else invalidism may result, or a diagnosis of psychoneurosis may affect the patient's entire outlook on life.

Strains, long forgotten, may have been the irritant initiating the pain cycle, and illness with concomitant vitamin and endocrine imbalance may so increase the patient's susceptibility to pain that the syndrome develops. The trauma may be slight and recurring, as in bowling or horseback riding; it may result from a single injury, such as slipping or falling down stairs or from a step-ladder; or it may occur after an operation.

Such patients have been referred to me for surgical relief of cervical herniated disk or for thoracic sympathetic interruption for angina.

A few internists have recognized the syndrome as neuralgia and have located trigger spots which intensify the pain upon compression.

Falls, a wrench of the left shoulder while holding on to the overhead strap when a streetcar is suddenly halted and prolonged illness after viral pneumonia are here listed as known causative factors producing pain in the left chest and arm, for which the patient has been treated for months or years as having a weak heart or angina pectoris.

In a series of thirty patients seen by me from 1948 to 1953, each has had repeated electrocardiograms which have been consistently negative, and many have taken glyceryl trinitrate tablets with or without temporary relief. A patient under diagnosis of angina pectoris, whose activities are restricted, is always fearful of coronary thrombosis and actually maintains a mild alarm reaction. Such stress, well described by Selye,[4] can readily progress to subclinical or even clinical signs of endocrine insufficiency with perceptible aging and an altered personality.

The pectoralis major muscle (Figs. 1 and 2) has origins on the medial half of the clavicle, at the costosternal junctions of the upper six costal cartilages and the aponeurosis of the obliquus externus abdominis muscle. These origins are not only at the clavicle and costosternal cartilages, but are also along the anterior surface of the rib-cage as far as the anterior axillary line. Trigger spots, therefore, are not limited to the costosternal region on the left side, but may be beneath the mammary gland. The pectoralis minor muscle has its origins from the second, third, fourth and fifth ribs near their cartilages and on the rib-cage. The trigger areas of the minor muscle are beneath the pectoralis major muscle and usually beneath the breast. Trigger points may be found at the insertion of the pectoralis major into the bicipital groove and the capsule of the shoulder joint, and at the insertion of the pectoralis minor to the medial border and upper surface of the anterior half of the coracoid process and into the tendon of the coracobrachialis and biceps muscles.

In definite cases the location of these irritative trigger points has been such that a slight movement, such as turning, arising from the bed and even sudden noise with resultant tensing of the body, has initiated the pain. Prolonged and complete rest in bed can ease this pain which will recur and continue for minutes or hours upon slight effort.

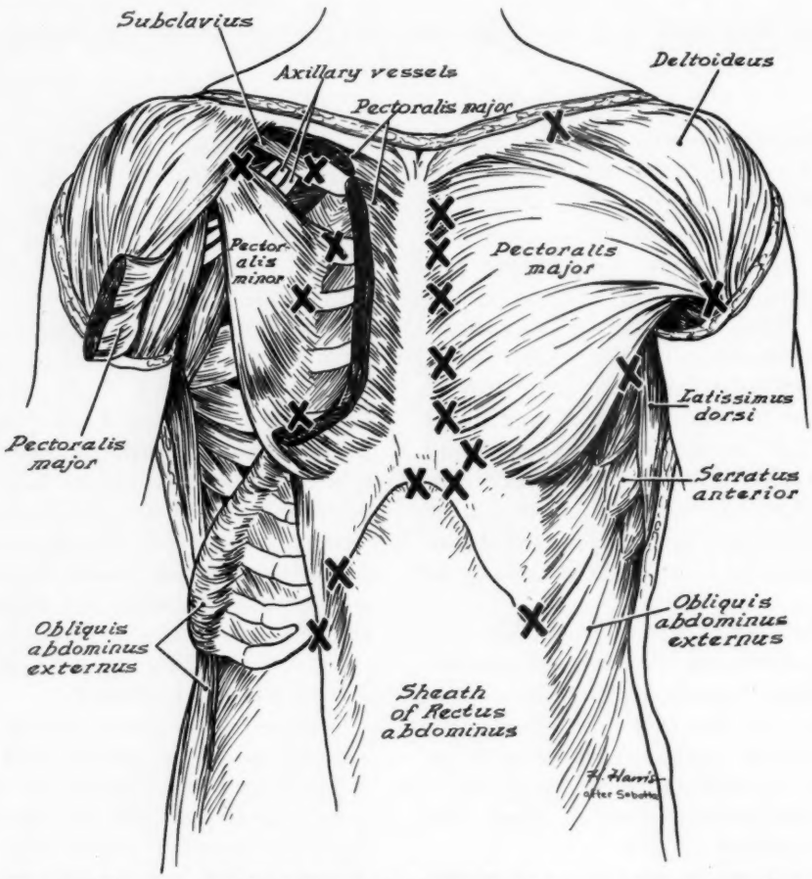

FIG. 1. The cross marks indicate the location of the usual trigger points. (Modified from Sobotta's Atlas of Human Anatomy. W. B. Saunders Co., Philadelphia.)

The majority of my patients with this neuralgia had consulted numerous internists; physical examination revealed no cardiovascular disease, electrocardiograms were repeatedly negative, and although their pain was unresponsive to cardiovascular therapy, the diagnosis of angina pectoris or tired heart was offered on the basis of their symptoms alone. Their unremitting pain over the years then prompted them again to seek relief in lieu of a life of invalidism and anxiety to their relatives.

Success in injecting trigger points with 2 per cent procaine hydrochloride will depend upon the most accurate identification and painstaking infiltration of the sensitized areas. Such a trigger area is first located by making pressure with the rounded end of a ball pen and then its tip, progressing tangentially toward a central point of maximum sensitivity and must be rechecked several times, always with pressure in the same plane and direction. Then the spot is marked so that when a No. 25 needle with a very short bevel is introduced, exactly the same direction is followed as with the pen. The deposition of the 2 per cent procaine is begun just beneath the skin and made gradually deeper until all trigger spots have been reached. Usually from 2 to 5 cc. are injected in each spot and not more than 40 or 50 cc. are injected at a visit. As the needle-point touches the trigger spot, a momentary flare of typical pain occurs which fades as the procaine is injected. After the injection the spot must be firmly compressed with the finger for five

minutes or more to prevent hemorrhage in the path of the needle. A hematoma at the site of the infiltration will vitiate the success of the injection, so that patients who bruise readily are given ascorbic acid, 500 mg. or 10 mg. of a vitamin K preparation intramuscularly prior to treatment even though their blood studies are found to be normal.

No patient receives an injection of procaine, no matter how small the amount, without first receiving a barbituate which reduces the toxicity of the procaine about five times. Often ½ gr. of phenobarbital by mouth twenty minutes before injection will suffice. Patients who are known to be sensitive to procaine receive ½ gr. of phenobarbital orally three hours, two hours, one hour and one-half hour before treatment. An ampule containing 2 gr. of sodium phenobarbital is always available, half to be given intramuscularly if a patient should react unfavorably to the procaine. I have found procaine in the concentration of 2 per cent to be the most effectual for permanent relief.

Two weeks or more after subsidence of the initial soreness, other "masked" trigger spots not at first in evidence can, if necessary, be located and injected.

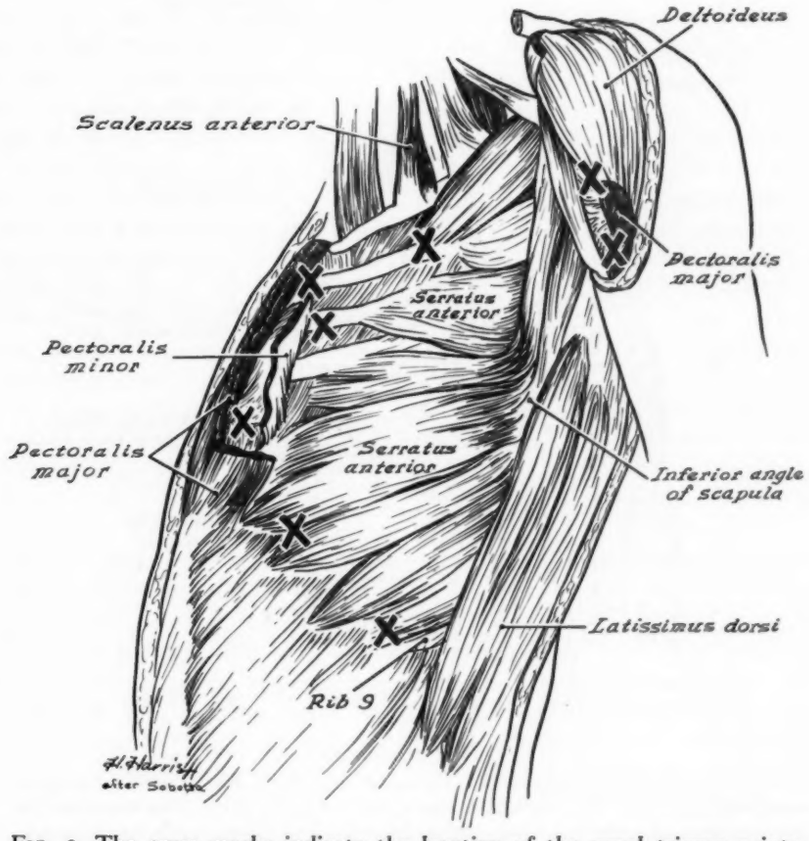

FIG. 2. The cross marks indicate the location of the usual trigger points. (Modified from Sobotta's Atlas of Human Anatomy. W. B. Saunders Co., Philadelphia.)

In this group of so-called cases of angina pectoris, some of the patients have pain under the left breast in the anterior axillary line and down the left arm and forearm. Consideration of a ruptured intervertebral disk[5] is eliminated by the complete absence of any of the diagnostic signs of a disk. The anterior scalenus syndrome must be kept in mind as pressure over this muscle behind the clavicle often accentuates the pain, and sleeping on three pillows,[6] as a conservative measure will give considerable relief in a few cases. In others, however, procaine injections into the potential bursa and loose areolar tissue between the spinous processes of C6-C7, of C7-T1 and T1-T2 with the needle-point directed just to the transverse

arch or ligament (Fig. 3) will cause radiating pain in one or both upper extremities, identical with their complaint. When, by these interspinous injections, the relief is not complete, additional trigger points will be found on the ribs in the axillary line where the pectoral

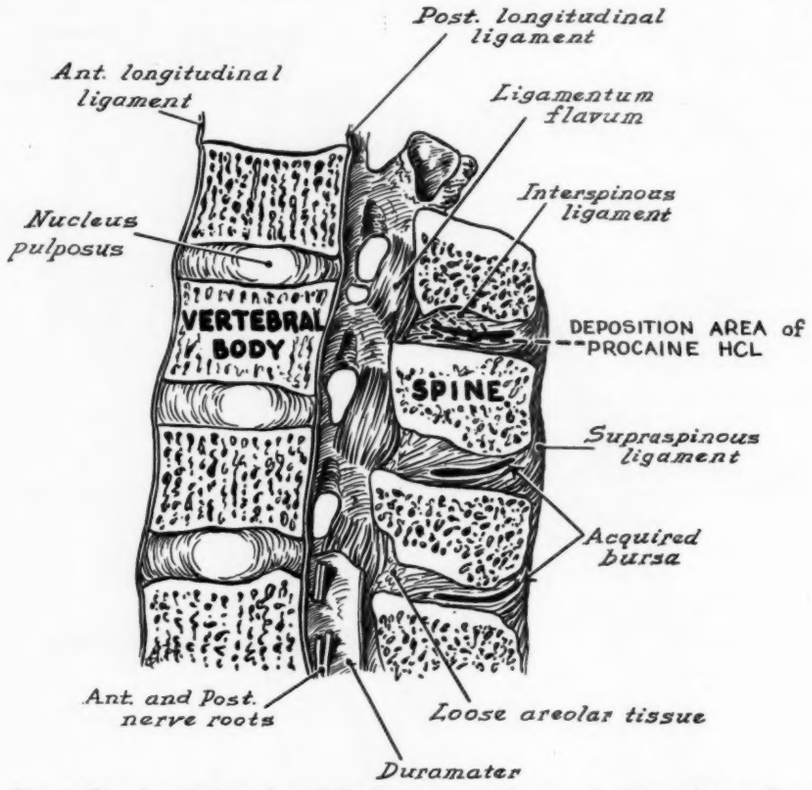

FIG. 3. Showing the location of the ligamentum flavum, the interspinatus ligament with its bursa. The needle for procaine injection penetrates between the spinous processes into the interspinous ligament and bursa, down to the transverse arch or the ligamentum flavum. (Modified from J. C. B. Grant's Atlas of Anatomy. Williams & Wilkins Co., Baltimore, 1947.)

attachments arise, or at the insertions of the pectoralis major and minor, or in the subdeltoid bursa.

Of thirty patients seen, twenty-three have remained free of pain after injections for eleven months to five years. Three were improved but did not return for further treatment. Two were helped temporarily, and wished no further treatment. Two never responded to follow-up letters.

A brief summary of the history and results in three patients of this group follows.

CASE REPORTS

CASE 1. Mrs. Z. S., age forty, had had pain in the left chest for nineteen months, reportedly starting with neurasthenia which followed a respiratory infection. The history revealed, however, that the pain had occurred earlier, with a fall from a step-ladder, and has been present ever since. This pain which had always been substernal and on the left was sharp and brief at first; later it had become a more pronounced ache, more severe when nervous or fatigued from household work and improved by rest in bed. A diagnosis of angina pectoris had been made despite several negative electrocardiograms. Electrocardiograms and roentgenograms for heart size and shape were reported to be normal, as were the pulmonary and general physical examinations. At least three very tender regions were located on the wall of the left chest and bilateral hypothenar erythema was noted.

The patient appeared thin, old and graying. On March 5, 1951, the trigger areas on the left costosternal margin were injected with 2 per cent procaine, as were additional trigger points on the left tenth rib below the nipple and

between the spinous processes of thoracic one to six; complete relief of pain resulted. A well balanced vitamin formula with minerals was administered daily with suprarenal cortex by mouth every three days.

On a return visit three weeks later the patient was entirely free of her "heart" pain, the tired, gaunt look was gone as was an eczematous rash on her forearm. The hypothenar erythema was improved. She had found several "masked" trigger points on the left costosternal margin as well as on the left fourth rib beneath the breast that were sensitive to pressure and were injected at this time.

Two months after the first injection she felt completely well and had resumed all activities. Although she had had no "heart" pains, there were three additional tender spots which were injected. A month later she looked and felt fine. She has remained well and a letter written October 2, 1953, states, "I'm fit as a fiddle . . . my chest is as good as ever. I've never had any trouble since you injected me."

Case II. Mr. R. Y., age fifty-three, was referred to the medical wards for either coronary disease or dissecting thoracic aneurysm, although roentgenograms of the chest and electrocardiograms were normal. He had had a constrictive feeling in his chest on exercise or excitement for the past six months, with a sharp, throbbing pain over his heart. Rest and glyceryl trinitrate had relieved the pain temporarily; but when he was startled by the banging of a door or the ring of the telephone, the pain recurred. There was no sign of arteriosclerosis and electrocardiograms at rest and after exercise were normal. It was believed that he might have angina pectoris with some neurotic overlay, but his complete disability did not seem fully explained. A sympathetic ganglionic block of thoracic one to six on the left was suggested. When I examined the patient on March 4, 1951, the minor effort of raising up on his elbow or turning to get out of bed caused pain which he described as a tight band under his sternum. A number of trigger points were found on the left costosternal border which when pressed upon, and also when the needle was introduced, exactly reproduced the constrictive feeling of his complaint. When his head was bent forward, a longitudinal ridge from occiput to C7 spinous process was seen. This "bowstring" had been caused, as found incidentally by me at former operations, by a trauma and tearing of the ligamentum nuchae from its attachments to the cervical spinous processes. Sensitive spots between C3-C4 and C6-C7 were injected, with referred pain down his left arm. Further search and efforts on his part now failed to reproduce pain and he was dismissed the next day.

His history revealed that late in August, 1950, while working as a game warden, he was loading a second large buck into the car which required a tremendous strain to lift it on top of the first one. Two days later the constrictive feeling, substernally, prevented him from working. Since its onset there had been a gradual decrease in the amount of effort or exertional tension required to bring on the tightness.

When seen at the clinic three months later, in June, 1951, he had not been back at work, as there were good days without constrictions and days when the slightest exertion or excitement caused tightness. Six more trigger points were injected at the left pectoral origins, a point on the zyphoid, and between the spinous processes of C4-C5, C5-C6, C6-C7 and C7-T1, also in the right subdeltoid and over the right second rib near the sternum. The patient resumed work five months after these injections, but stopped after four months when his case was considered by a commission. He was awarded permanent disability compensation, has not done any work and does not plan to return to the clinic as he is taking "angina" pills for his nervousness. When last seen on January 19, 1954, he stated that the trigger spots were gone and the "angina" pain was different from what he had in the hospital.

Case III. Mrs. J. S. B., age fifty, had had anginal pains for over ten years. They began after a fall down stairs which was not considered a contributing factor. The pains were precordial and down the left arm, more noticeable when fatigued or upset and not helped by glyceryl trinitrate. The employment of multivitamins seemed to have improved her condition. Electrocardiograms were always normal, but she was warned to spare her heart. When last seen on December 17, 1953, she had been relieved of pain since April 14, 1951, when trigger points were injected with procaine along the origins of the left pectoral muscle to the clavicle, to the second, third, fourth and fifth and sixth ribs under the breast, and in the left subdeltoid region.

SUMMARY

Cases of atypical neuralgia or post-traumatic pain syndrome involving the chest that resemble angina pectoris are unusual and few in number compared with cases of true angina pectoris.

One is cognizant always of the fact that remote disease of the viscera, in particular the heart, can give rise reflexly to trigger areas in the musculofascial regions, but in each of this series of thirty atypical cases under discussion visceral disease has been repeatedly proven absent.

The main source of irritation in the chest is due to sprains or strains of the origins of the pectoral muscles, and at times of the trapezius, deltoid and supraspinatus muscles.

Pain down the arm can be relieved by procaine injections into the potential bursa between the spinous processes of the lower cervical and upper thoracic vertebrae.

The prolonged anxiety to the patient under a mistaken diagnosis of angina pectoris, whose activity is restricted in constant fear of a coronary thrombosis, and the exhaustion from pain precipitate and maintain an alarm reaction which progresses in severity over many years.

These patients develop subclinical and clinical signs of vitamin and endocrine insufficiency, and age perceptibly, often showing marked changes in personality.

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Non-meckelian Diverticula of the Jejunum and Ileum

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IN 1807 Cooper[3] first described jejunal diverticula in the English literature. This was an autopsy finding. In 1881 Osler[7] made a similar report in the American literature, also as a result of an autopsy finding. It was not until 1906, however, that Gordinier and Sampson[5] reported this finding in a living patient, a finding made at laparotomy in a patient with intestinal obstruction. Fourteen years passed before the diagnosis of jejunal diverticulum was made preoperatively by x-ray and confirmed at operation. While the number of these reported cases exceeds 300, only a very small number has been subjected to laparotomy.

Incidence. In 1936 Rosedale and Lawrence[10] decided to investigate the incidence of jejunal diverticula as accurately as possible at autopsy. Using the technic of insufflating air into the jejunum under moderate pressure, they found four cases of jejunal diverticula in 300 autopsy patients, an incidence of 1.33 per cent. In 1935 Rosedale[9] studied the records of 5,000 routine autopsies and had found only three instances of this disease, an incidence of 0.06 per cent. Apparently at autopsy, when a particular search is made for this lesion, it will be found more than twenty-two times as frequently as when necropsy is general and routine. Edwards,[4] in a survey of the protocols of 2,820 autopsies, noted the presence of jejunal diverticula in nine cases, an incidence of 0.3 per cent. Furthermore, when he surveyed the x-ray reports in 11,362 barium meals, only seven cases were found, an incidence of 0.06 per cent. Rankin and Martin[8] purposely looked for this lesion in x-ray surveys of the small intestine and found one case in 479 examinations or 0.22 per cent. However, when barium meal examinations were made in a routine manner only, they noted its report only once in 25,000 instances or 0.004 per cent. Orr and Russell[6] reviewed 2,161 barium meals and found nine cases, an incidence of 0.42 per cent. No mention is made whether or not special small intestinal studies were made to find these lesions. Wilkerson and Coffman[13] could not find any cases in the records of the Maryland General Hospital, Bon Secours Hospital and the South Baltimore General Hospital. The records of the University of Maryland Hospital yielded two cases, although the number of records searched is not mentioned. In Houston the diagnosis of non-meckelian diverticula of the jejunum and ileum has been made nine times in three of the four major hospitals in the last ten years, during which time there was a total of 462,725 admissions.

The disease shows no preference for sex but customarily is noted in those of middle or upper years.

Site. In the series of Benson, Dixon and Waugh[2] (122 cases) the jejunum was involved alone in 100 cases, the ileum alone in seventeen cases and in five instances diverticula were found along the entire small intestine. Edwards[4] noted eleven cases in which the jejunum alone was involved and one involving only the ileum. In the reported series of Baskin and Mayo[1] the jejunum alone was involved in seventy-six cases, the ileum alone in four and in seven cases both jejunum and ileum were involved. In fifty-nine of these eighty-seven cases the colon itself was studied by barium enema, at laparotomy and by sigmoidoscopy, and colonic diverticula were noted in 44.1 per cent of the cases. Bockus indicates that colonic diverticula occur in 5 to 10 per cent of the general population.

Pathology. Typically the disorder is represented by herniation of the mucous membrane of the affected part of the bowel between the fibers of the muscularis at the site where a blood vessel penetrates the wall. In the vast majority of cases this is on the mesenteric side of the intestine. In a small number of instances diverticula may originate in the extramesenteric portion of the bowel circumference. In these instances a large vessel will usually be

present, causing a separation of the muscle fibers at the site of the diverticulum. Edwards clearly shows this relationship of these diverticula to blood vessel penetration of the bowel wall as being so constant as to refute the possibility of coincidence. This point of emergence of the diverticulum he has designated as the "locus minoris resistentiae." A deficiency in the continuity of the muscular coats of the intestine results when the blood vessels pass through them. As the vessel passes through the intestinal wall, it is at an angle from the vertical estimated to be about 55 degrees; and its passage through the muscular coat itself is oblique. With an increase in the intraluminal pressure at this point of "locus minoris resistentiae," a wedge is created by the mucous membrane as it is pushed between the blood vessel and the adjacent muscularis of the wall. As pressure is constantly applied and resistance is unable to oppose it, the diverticulum comes to be a permanent entity. As it progresses it proceeds along the path of least resistance, the blood vessel. As a result it pushes into the mesentery, not exactly into the middle but along one side of it. This is because the blood vessel to the bowel, the so-called terminal vessel, running up through the midline of the mesentery, divides just as it reaches the bowel. These two branches clasp the bowel wall within themselves, each one supplying that part of the intestinal wall into which it runs. Each penetrates the bowel just off the mid-point of the arc of the bowel circumference included by the two walls of the mesentery. It is for this reason that the herniation of the mucosa, following the path of the blood vessel which pierces the muscularis, is situated asymmetrically within the mesentery. This pathogenesis clearly explains why these diverticula lack a muscular coat and consist of the mucosa and submucosa covered by serosa only. The congenital type, best exemplified by Meckel's diverticulum, is invested by all the normal layers of the intestinal wall.

Clinical Features. Orr and Russell have presented a practical classification of this disease.[6] For that reason it may be used as a guide for treatment in this type of case. Patients with non-meckelian diverticula of the jejunum or ileum may be placed in one of three groups at any particular time.

Group I consists of those patients who exhibit no symptoms at all. This group is very likely numerically greater than either of the following groups, and probably greater than both of them combined. When an autopsy study was made, very pointedly looking for diverticula of the jejunum and ileum, an incidence of 1.33 per cent was found.[10] It is proper to assume that a large number of these are without symptoms for the duration of the individual's life. In this group a diagnosis of this disease is made only in the performance of routine study with a barium meal and much less frequently during the course of laparotomy.

Group II includes those patients who have chronic indigestion or dyspepsia. In the effort being made today to diagnose gastrointestinal disease as accurately and as early as possible, the barium meal is a standard diagnostic procedure. Its most frequent use arises in those individuals complaining of a vague, low grade, abdominal disability, the cause of which is not clinically clear. When the barium meal study is customarily performed, great care is given to examination of the stomach and duodenum but little to a small intestinal study unless it is specifically requested, and this is done only very occasionally. Usually the first part of the examination is terminated before the barium has had time to fill any jejunal or ileac diverticula which may be present, and the five to six-hour film may miss them because they have had time to empty. However, as frequently occurs, no cause can be found for the vague, chronic abdominal discomfort felt by the patient. Since Rosedale and Orr found that 1.13 per cent of consecutive patients subjected to autopsy examination had jejunal diverticula, it is not unreasonable to assume that in each group of 10,000 people 113 will have this finding. In Group I it was pointed out that it is altogether likely that the majority of people with jejunal diverticula never did have symptoms and would never appear for medical attention on that score. It can also be reasonably assumed that much more than a majority of people never appear before a physician for a barium meal study. If these postulates are acceptable, it follows that of all those people seeking medical help because of dyspepsia, a greater number than 1.13 per cent have jejunal diverticula causing or contributing to their abdominal discomfort. If, in addition, we refer to the series of Benson, Dixon and Waugh[2] and note that diverticula of the ileum alone occur about 16 per cent as frequently as

diverticula of the jejunum and jejunum and ileum, the incidence of non-meckelian diverticula of the jejunum and ileum in patients complaining of dyspepsia will be greater than 1.13 per cent. A search for this entity when no gastric, duodenal, biliary, pancreatic or colonic cause for the patient's complaint is found in the course of a barium meal examination will reveal the presence of jejunal and/or ileac diverticula responsible for the complaint in a higher percentage of patients than is now being recognized. It must be borne in mind that very often no abnormal radiologic appearances are produced, even when special methods are adopted to demonstrate the small intestine. Therefore a negative examination does not necessarily exclude the condition but does make its presence less likely. Like diverticula anywhere in the bowel, jejunal or ileac diverticula may become inflamed as a result of incomplete emptying. Such inflammation certainly can cause indigestion of varying intensity. The neck of the sac may close off because of the swelling and, in turn, this may lead to formation of a concretion. Symptoms are vague and are presented as gas, belching, flatulence or noisy stomach, besides definite abdominal discomfort of varying intensity. When the latter becomes progressively more severe and incapacitating, Group III is encountered.

Group III consists of those patients designated as having "acute complications" of the diverticula. It is in this group that most of the patients with non-meckelian diverticula of the jejunum and ileum are diagnosed as a result of laparotomy. The complications of acquired diverticula of the jejunum and ileum which usually require surgery have been classified by Walker[12] in the following manner: (1) Acute diverticulitis causing generalized peritonitis, localized abscess, adhesions and acute intestinal obstruction (these complications may or may not be associated with actual perforation); (2) concretion formation which may cause intestinal obstruction; (3) hemorrhage; (4) traumatic rupture; and (5) volvulus.

Diagnosis. In a patient with chronic indigestion in whom the more common forms of gastrointestinal disease have been excluded, a study of the jejunum and ileum would appear to be indicated, with diverticula borne in mind. In order to see the barium filling a jejunal or ileac pouch, the meal should be followed at half-hour intervals.[6] When a diverticulum is demonstrated fluoroscopically, tenderness elicited by palpating it would appear to indict it as the cause of the dyspepsia in the absence of any other gastrointestinal disorder. X-ray study is the only absolute means of diagnosing this pathologic condition short of laparotomy.

Treatment. Those diverticula found accidentally and obviously causing no disability require no treatment. The patient should, however, be informed of their presence and the prognosis outlined so that if any of them ever become a factor to be considered in a differential diagnosis, the physician consulted may receive this information from the patient. When the case falls into Group II (chronic dyspepsia), treatment is of course medical until such time when it is obviously inadequate. Medical treatment can be made effective if it is kept in mind that the only diverticula causing complaints and not requiring surgery are those which empty poorly because of an inflammatory process which constricts the outlet, thereby eliminating good drainage. The usual measures of low residue diet and antispasmodics are frequently used, but more specific medical measures directed toward resolution of the inflammation with resultant widening of the outlet and adequate drainage from the lumen of the diverticulum would appear to be more precise. For this reason the use of a bland, low residue or liquid diet combined with neomycin and sulfasuxidine® for their local effect, with or without the addition of penicillin for the systemic effect, would appear to offer far more specific effect than not using them at all.

Surgical treatment of this disorder varies in degree from simple resection of the sac to resection of a variable part of the involved bowel. When laparotomy is elective, it offers little risk in itself. It is rather the constitutional defects, such as degree of obesity or the cardiac status of the patient himself, which may be the factor in deciding on laparotomy when medical treatment has been found wanting in patients bordering between Groups II and III and when no acute surgical condition of the abdomen is present.

When these diverticula are responsible for an acute abdominal emergency, the correct preoperative diagnosis will be made very infrequently. Shutkin,[11] however, reported a case in which the diagnosis was made by barium meal study during an episode of jejunal diverticulitis. This patient responded to medical care

but had a recurrence less than three weeks later; laparotomy was performed. Resection of the perforated diverticulum resulted in an uneventful convalescence. Nevertheless, in the great majority of cases the cause will be found only as a result of exploration. Unless it is considered, diagnosis will never be made preoperatively or suggested as a possibility in an acute abdominal emergency. These factors are well illustrated in the following case report.

CASE REPORT

This obese fifty-five year old man noted the sudden onset of midline suprapubic pain one morning. This was soon accompanied by anorexia and nausea with supervention of two episodes of vomiting that afternoon. During the day the patient had a normal bowel movement. Although accustomed to a hard day's labor, he was unable to continue his work that day. On returning home he went to bed. The pain now spread laterally to both sides of his abdomen but was somewhat more pronounced in the right lower quadrant. A self-given enema resulted in a good return. Shortly after the onset of the pain he noted the beginning of abdominal distention. The distention progressively increased and the patient vomited twice again during the ensuing night. The patient, a stoic individual, hoped that his distress would abate without resorting to medical care. A restless night was passed in his home; during its course he slept fitfully. The next day failed to bring the hoped-for relief. Instead he noted the slow but continuing abdominal distention and the increase of pain in the lower half of the abdomen with gradual ascension to the upper half. Medical help was therefore summoned.

The patient was hospitalized for observation. On admission the usual routine work-up was done and a scout film taken of the abdomen. Examination of the patient revealed an obese white man who was obviously ill but not distressingly so. The sensorium was normal and prompt, and accurate response to questions was forthcoming without hesitation. In the search through the history for more precise facts by several questioners the patient never altered the facts as given in the original history. The abdomen was pronouncedly distended but was not taut. No board-like rigidity was noted. The examining fingers could be sunk into the abdomen without undue pressure. On deep pressure, tenderness was elicited in the lower half of the abdomen but it was more pronounced in the right lower quadrant, most particularly over McBurney's point. The patient had had a right inguinal hernia repair twenty years previously, at which time he had been told that his appendix had been removed. No masses could be felt. The liver and spleen could not be palpated. Abdominal auscultation revealed active peristalsis. The lungs were clear. No evidence of hernia could be found. Rectal examination revealed diminished tone of the rectal sphincter and a dilated ampulla. The scout film of the abdomen was reported as follows: "A scout film of the abdomen shows moderate gaseous distention of several loops of the small bowel which are arranged in a step-ladder pattern suggesting intestinal obstruction. There is also a moderate amount of gas distributed irregularly throughout the colon. This would indicate that the obstruction, mechanical, probably is not complete. Findings similar to these can be seen in elderly bedridden patients and also in some acute abdominal conditions such as ureteral stones." The white blood cells numbered 9,800 with 80 per cent polynuclears and a shift to the early forms. Urine was normal.

With the acquisition of these data and still unable to make an etiologic diagnosis, the patient was repeatedly examined over the next two hours. History and findings remained unchanged except for a gradual diminution in the intensity of the peristaltic sounds.

Within the previous two months another patient had been seen with a history of right inguinal hernia repair thirty years before, with appendectomy through the same incision. This patient was found to have generalized peritonitis secondary to a ruptured gangrenous appendix. The possibility was entertained that the same situation could be present here. The second possibility strongly considered was that of a ruptured diverticulum of the colon. Nevertheless, the only preoperative diagnosis that could be entertained convincingly was peritonitis secondary to a ruptured viscus. On this basis the decision to perform an exploratory laparotomy was made.

Just proximal to what was estimated to be the middle of the jejunum were four ballooned-out areas of the mesentery adjacent to and hugging the intestine. The largest of these was 4 cm. in diameter and the smallest 1 cm. They

were all located on the caudad side of the mesentery. Moderately inspissated, purulent exudate was plastered against the tissues in this area with most of it concentrated on and around the largest of these saccules. No active leakage could be seen emanating from it. Some of the exudate was easily-wiped away, revealing a 2 mm. opening in the wall of the sac from which a small amount of frothy, thin fluid soon oozed. Invaginating the wall of this sac with the finger indicated an opening into the intestine large enough to admit the finger. The same procedure was followed in regard to the other three diverticula. In each case, as in the perforated one, a large communication into the intestine was found. Treatment consisted of removal of the perforated diverticulum and closure of the opening in the bowel wall. This same treatment might well have been extended to the other three diverticula but was not; they were permitted to remain undisturbed. Anatomic closure of the abdominal incision was performed without drainage. The patient's postoperative course was uneventful.

The pathologic specimen was the wall of the ruptured diverticulum of the small bowel. Grossly, the specimen consisted of a piece of brown-gray, moderately firm tissue measuring 2 by .7 by .5 cm. Microscopically, sections showed adipose tissue with a smooth outer lining of serosal type. The adipose tissue was heavily infiltrated by a purulent, inflammatory exudate, and necrotic fibrin was also present. In some areas of the section small foci of fat necrosis were present. Careful examination of the section failed to reveal any parasitic agent. The microscopic findings were compatible with that found in a ruptured diverticulum. Diagnosis was rupture of the wall of the diverticulum.

It is possible that had no surgical intervention taken place, the patient might have gone on to spontaneous cure if the closure achieved by the purulent exudate had been maintained. This, however, is problematic. While the preoperative picture was not textbook clear, sufficient indication existed making laparotomy mandatory. The prompt postoperative response indicated that this was the correct procedure.

COMMENTS

The presence of diverticula of the jejunum and ileum as a cause of chronic dyspepsia receives very little consideration as the cause of this complaint in the absence of the more common forms of gastroenterologic disorders. One of the reasons for this is its lack of consideration as a possible cause of such complaint. In turn this is due to the infrequency with which the diagnosis has been made. It has been pointed out that the likelihood of its clinical appearance may exceed 1.3 per cent. This is thought to be a rather conservative estimate. It is apparent that the cause of dyspepsia is never ascertained in a large percentage of patients, appearing with the complaint of dyspepsia, in whom barium meal studies are done without the inclusion of a small intestinal study. These patients are placed on a regimen of bland diet and antispasmodics with gradual resolution of the complaints in some while the remainder undergo a somewhat later spontaneous cure. This undiagnosed group may be conservatively estimated to range from 25 to 33 per cent. There need be very little doubt that a fraction of this number have as their disorder diverticulitis of the jejunum or ileum or both. A serious attempt by one competent to do a study of the jejunum and ileum with a barium meal will most certainly result in more frequent diagnosis of diverticula of the extraduodenal portion of the small intestine.

SUMMARY

Dyspepsia is a term used to define a variable degree of intra-abdominal discomfort when no specific reason for the disability can be found. As diagnostic accuracy rises, so inversely, does the number of cases of abdominal complaints diminish under this catch-all, diagnostic, mean-nothing term.

Although diverticula of the jejunum and ileum have been discussed in the literature for almost 150 years, consideration of it in the differential diagnosis of chronic dyspepsia is rarely entertained. Its occurrence accompanying other gastrointestinal disorders or its presence alone as the cause of chronic dyspepsia is far more frequent than is revealed in hospital discharge records. When the more common forms of gastroenteric disease are eliminated, consideration of and search for this disease process will indicate its presence more often than is now being done. The basis for making this diagnosis has been discussed.

A brief outline of the medical and surgical

treatment of this disease has been given. The infrequency of this diagnosis in hospital discharge records is unwarranted.

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Pylephlebitis and Liver Abscess

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Pylephlebitis, first described by Waller in 1846,[8,10] is a disease characterized by suppurative thrombophlebitis of the portal vein, with extension into its intrahepatic branches, and by the formation of single or multiple liver abscesses mainly in the right lobe of the liver. Although it may follow suppurative disease anywhere in the portal drainage system, and has even been reported from infected hemorrhoids,[2,13] it has been associated with suppurative appendicitis in the majority of reported cases. It has been suggested that the suppurative process might reach the portal area through lymphatic drainage,[3,4] but most cases can be explained by direct extension[5] involving the veins of the mesoappendix, ileocolic vein and superior mesenteric vein in turn, or by blood-borne emboli.[9] Regardless of the site of the suppurative disease, the responsible organism is most frequently Bacillus coli, with Streptococcus hemolyticus and Staphylococcus aureus following in that order.[13]

The present study is based on a series of seventeen cases of pylephlebitis collected from three Cleveland hospitals: five each from University Hospitals and Crile Veterans Hospital, and seven from Cleveland City Hospital. The incidence of appendicitis as the source of suppuration was not quite as high as in most reported series. Ten cases, or 59%, were preceded by acute gangrenous or perforated appendicitis while the antecedant diseases of the other seven cases, or 41 per cent, were divided equally between the peritoneal and pleural cavities. (Table 1.)

Of the five patients at University Hospitals, three had had appendectomies for acute appendicitis, one acute and two perforated. They had been operated upon in a series of 6,014 appendectomies for acute appendicitis thus giving an associated incidence of pylephlebitis of 0.05 per cent. This compares with an incidence of 0.09 per cent reported by Ochsner in 5,293 cases of appendicitis. There were 659 perforated appendices in the University Hospital series giving an associated incidence of pylephlebitis of 3.0 per cent. At autopsy Petren found pylephlebitis was the cause of death in 5 per cent of 1,340 patients dying from appendicitis.[15]

Table 1
Source of Pylephlebitis

	No. of Cases
Appendicitis	10
Acute	4
Acute perforated	5
Acute gangrenous	1
Ulcerative enterocolitis	1
Pulmonary abscess	1
Perforated carcinoma, stomach	1
Emphyema with pneumonia	1
Perforated duodenum	1
Acute pancreatitis	1
Carcinomatosis	1
Total	17

The present series is much too small to permit valid conclusions, but it does present some interesting material. Pylephlebitis occurred in all decades except the first two and was most prevalent in the third when appendicitis was most common. (Fig. 1.) There were five Negro and twelve white patients, demonstrating no racial influence. Prior to 1946 there were eleven cases with eleven deaths, a mortality rate of 100 per cent. Since 1946 there have been six cases with three deaths, or a mortality rate of 50 per cent. Autopsies were obtained in all but one case which had been confirmed by operation and in which the clinical course had been identical to the autopsied cases. The three patients who lived had adequate laboratory data and clinical and operative findings to justify the diagnosis.

The diagnosis of pylephlebitis and liver abscess was usually very difficult to make clinically and was almost always belated if

made at all before autopsy. The history of appendicitis was frequently so atypical that it was overlooked entirely. In three of the cases of appendicitis, the fever and shaking chill preceded the nausea and abdominal pain by two to six hours. In two other cases of appendicitis, the chill followed the onset of nausea and abdominal pain by six to twenty-four hours, and in one it occurred six weeks after operation for gangrenous appendicitis. This is in accord with previously reported cases which followed appendectomy by six and seven weeks.[3,4] In analyzing 2,841 cases of acute appendicitis, Colp found an incidence of 6.8 per cent of preoperative chills, but pylephlebitis was usually preceded by multiple and protracted chills.

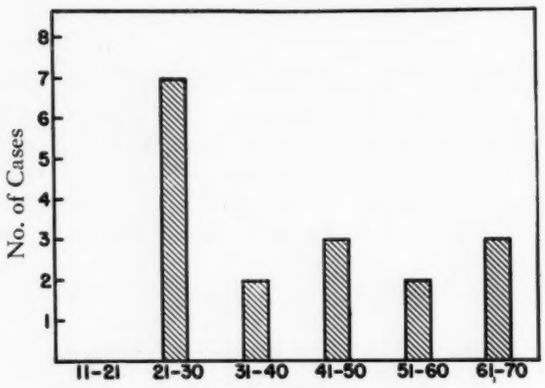

FIG. 1. Prevalence of pylephlebitis by decade.

The fever of pylephlebitis was septic after a dramatic onset with chills and ranged up to 40 or 41°C. There was an occasional decrease in this fever with a change in antibiotics or the use of repeated blood transfusions, but it usually persisted until the death or recovery of the patient.

Nausea was an evident feature of the disease but might have been associated with the primary disease which caused the pylephlebitis. Many of the patients had diarrhea, but the character of the stool was not diagnostic and in no case was it bloody or excessively mucoid.

Jaundice was an important clinical manifestation of the disease and a great aid in its diagnosis.[3,4,6,8] It was present in twelve of the seventeen cases but was usually mild with icterus indices of from 19 to 25. Three patients had marked jaundice, however, with icterus indices of 50, 55 and 60, respectively. Jaundice usually followed the chills and fever by a few days and persisted until the death or recovery of the patient. There was an associated positive cephalin flocculation, and an elevated thymol turbidity in the six patients tested; in three of the five patients who had serum protein determinations made there was a reversal of the A/G ratio.

The blood culture was positive in nine of thirteen patients tested. Five of the nine had negative cultures early in the disease but these subsequently became positive. The causative organisms were Escherichia coli in three, hemolytic streptococcus, hemolytic staphylococcus aureus and Streptococcus viridans in one each, and mixed cultures in the other three of Staph. aureus, Esch. coli and streptococcus; nonhemolytic streptococcus and staphlococcus; and Esch. coli and Aerobacter aerogenes. It appeared that the blood culture did not become positive until liver abscesses had formed with secondary seeding of the blood stream relatively late in the disease.[3-5]

The liver was recorded as enlarged clinically in five of the seventeen patients. This may not represent a true incidence as the liver is usually enlarged at some time during the course of the disease. The spleen was enlarged in four patients two of whom subsequently were found to have splenic vein thrombosis. Moderate ascites was present in two patients, one with acute appendicitis and the other with perforated carcinoma of the stomach. Ascites is rarely a prominent clinical finding with pylephlebitis.[3,4,8]

The course of pylephlebitis and liver abscess was prolonged lasting from nine to seventy-three days, with an average of thirty-two days in the fatal cases. The three patients who lived were hospitalized thirty-nine, eighty-six and 200 days, respectively.

Treatment before the advent of the antibiotics was chiefly directed toward the underlying disease and consisted of appendectomy, drainage of the appendiceal, subphrenic or other abscesses where indicated, and supportive treatment. Ligation of the ileocolic, superior mesenteric and even portal veins was given up in general as too hazardous after some early success with its use.[3,4,8] Following the availability of potent antibiotic drugs, the natural history of the disease seemed to have been altered.[1,11,12,14,18] Three patients survived in the present series and the average survivial of the three who died after 1946 was almost doubled.

A variety of the antibiotics and sulfadiazine were employed, with penicillin combined with streptomycin being favored unless sensitivity tests run on organisms obtained from the blood stream or an abscess cavity indicated a different choice. Three of the six patients treated since 1946 including two of the survivors were treated with 50 mg. of heparin intramuscularly every four hours. It was believed that this drug was contraindicated in the other survivor because of hereditary hemorrhagic telangiectasia of the gastrointestinal tract. The patient who died in spite of anticoagulant therapy did not receive it until the eighth week of his illness, two days before death. There was marked but temporary improvement in his clinical condition with a sharp drop in temperature.

Operative and autopsy findings in all cases revealed a suppurative focus somewhere in the portal drainage system with propagation of the process by direct extension to the portal vein and intrahepatic portal system in thirteen of the patients. In four, there was no involvement of the intervening veins so that it must be postulated that the disease spread by showers of septic emboli. The liver abscesses were usually small measuring 1 to 2 cm. in diameter. Large and confluent intrahepatic abscesses and subphrenic abscesses developed late in the disease providing that the patient survived long enough. Operative intervention for liver abscess proved effective only at this stage. Two of the patients who survived had large liver abscesses evacuated. The other survivor had drainage of an appendiceal abscess.

The six cases occurring since antibiotic drugs have been available are of special interest and are reported in some detail.

CASE REPORTS

Case 1. A twenty-seven year old Negress entered the hospital eight days after the onset of chills and fever which had been followed in one hour by vague upper abdominal pain. She had been mildly constipated for three days before coming to the hospital, and two days prior to admission she had become nauseated and had vomited. She had received three injections of penicillin during the eight days of her illness. On admission her temperature was 39.1°C., her lungs were clear by physical examination and her abdomen was distended. Spasm and rebound tenderness were present in the right upper and lower abdomen. No masses were felt. Laboratory findings included: a white blood count of 18,000; alkaline phosphatase 14.5; serum bilirubin, direct 1.4 mg. per cent, indirect 2.8 mg. per cent; thymol turbidity 4.7 units; cephalin flocculation 3+. A blood culture was positive for Esch. coli on admission. Following hydration she was operated upon with a diagnosis of empyema of the gallbladder. A perforated appendix with a carcinoid in its proximal third was removed. No observation was made about the appendiceal or ileocolic veins. The patient received terramycin intravenously and then penicillin and streptomycin. Her condition improved rapidly and antibiotics were stopped on the sixth postoperative day as her temperature was normal. On the ninth postoperative day chills and fever to 39.6°C. developed. Penicillin and streptomycin were reinstituted but were ineffective. Blood culture on the fourteenth day revealed Staphylococcus albus sensitive only to sulfadiazine and chloramphenacol. In spite of large doses of the latter drug, the patient's condition deteriorated. An attempt to drain a liver abscess on the twentieth day demonstrated no large abscess and she died at the close of the procedure. Autopsy revealed suppurative thrombophlebitis of the portal vein with multiple small liver abscesses.

Case 11. A twenty-eight year old white man entered the hospital with a history of chills, fever, headache and diarrhea of seven days' duration following drinking of raw water from a well near a privy. The onset of his illness had been marked by nausea and vomiting followed in several hours by a watery stool and shaking chill. He had been treated for five days at another hospital with sulfaguanidine, penicillin and streptomycin, as well as intravenous fluids. On admission he was acutely ill with a temperature of 104°F., a distended abdomen, palpable spleen, generalized lymphadenopathy and questionable rose spots. Agglutination to Eberthella typhosa was positive with both O and H strains to 1–1280. Cephalin flocculation was 4+ and thymol turbidity 6.3. There was an inversion of the A/G ratio. Blood culture showed no growth. X-rays revealed an inflammatory process in the right lower lung field and a laminated coprolith in the right lower quadrant of the abdomen. The patient ran a spiking fever up

to 106°F. with frequent chills. He was treated with emetine and diodiquin because of a positive complement fixation test for amebiasis. He also received large doses of penicillin, streptomycin and sulfadiazine without improvement. Exploratory laparotomy disclosed massive ascites and a huge spleen. Liver biopsy showed acute cholangitis with acute focal inflammation. The icterus index rose to 60 and the patient died on his sixty-sixth hospital day. Autopsy revealed thrombophlebitis of the portal and superior mesenteric veins with multiple periportal liver abscesses. The appendix was retrocecal and showed evidence of perforation in the vicinity of a large coprolith.

CASE III. A twenty-four year old white man was transferred from another hospital in the sixth week of an illness starting with mild abdominal pain and soon followed by malaise, chills and fever to 103 or 105°F. daily. A diagnosis of "intestinal flu" had been made and the patient had been treated with large doses of cathartics. On admission he was acutely ill, jaundiced, toxic, disoriented and extremely weak. There were generalized purpuric spots; the liver was palpable 5 cm. below the costal margin and the spleen was palpable. There was an anemia of 1,800,000 red blood cells, cephalin flocculation 4+, thymol turbidity 6.4 and an inversion of the A/G ratio. Blood culture was positive for Esch. coli with growth inhibited by aureomycin. X-ray of the abdomen showed a coprolith in the right lower quadrant. The patient was treated with sulfadiazine, penicillin and streptomycin without improvement. There was dramatic but temporary improvement after aureomycin, and blood cultures became negative. There was also marked temporary improvement after anticoagulant therapy with heparin was started two days before the patient died on his seventeenth hospital day. Autopsy revealed pylethrombophlebitis with multiple abscesses throughout the liver. There was an organizing periappendiceal abscess.

CASE IV. A fifty-six year old white man entered the hospital four months after operation for a ruptured appendix with peritonitis. Six weeks later epigastric pain, nausea and vomiting developed, and one month after that he commenced to run a fever from 100 to 102°F. which was treated with sulfa drugs at that time. Pain in the right upper back developed which was worse with cough and deep breathing. The patient was known to have congenital hemorrhagic telangiectasia of the gastrointestinal and respiratory tracts. On admission he was acutely and chronically ill, with a temperature of 103°F. There was an inflammatory process at the base of the right lung with a high right diaphragm, enlarged spleen, severe anemia with a red blood count of 1,500,000, and mild icterus with an icterus index of 21 rising to 25. The A/G ratio was reversed, cephalin flocculation was 4+, thymol turbidity 4.1 and blood cultures which were sterile at first grew Str. viridans sensitive to penicillin. The patient was treated with penicillin, seventy transfusions and surgical drainage. Several attempts were necessary before a liver abscess measuring 7 cm. in diameter and 4 cm. in depth, and a right subphrenic abscess culturing Proteus vulgaris, Staphl. albus and A. aerogenes were adequately drained. Convalescence was prolonged by a homologous serum hepatitis, and the patient was discharged with a draining sinus 200 days after admission. The sinus closed four years later after further drainage of subphrenic abscesses.

CASE V. A twenty-two year old white man entered the hospital with fever and abdominal pain which had started two weeks previously associated with headache, malaise and a chill followed by a fever of 103°F. The dull aching abdominal pain had localized in the right abdomen by the third day and had been associated with anorexia. The patient had been treated for amebic dysentery four years previously. On admission he was acutely and chronically ill with a fever of 104°F. The lungs were clear by physical examination but the right diaphragm did not descend normally. The liver was palpable 1.5 cm. below the costal margin and was tender. Blood cultures were sterile. Cephalin flocculation was 2+, thymol turbidity and A/G ratio were normal, and there was no jaundice. The patient was treated with emetine, but the complement fixation test for amebiasis proved negative. He ran a septic fever to 105°F., and received heparin, penicillin and streptomycin. The right diaphragm became high and fixed, and an area of tenderness and edema appeared over the right ninth rib anteriorly. Two weeks after admission, a two-stage drainage of a subdiaphragmatic and a large multiloculated liver abscess 2 cm. below

the surface of the liver was carried out. The pus cultured non-hemolytic streptococcus. Fever subsided slowly and the patient was discharged on the seventy-second hospital day. At appendectomy performed six months later there were many adhesions in the region of the appendix and sections showed healed appendicitis.

CASE VI. A twenty-six year old white man was admitted to the hospital with severe abdominal pain and loose watery stools on the tenth day of an illness starting with nausea and vomiting. Colicky pain in the right upper and lower abdomen had developed twenty-four hours after onset and had localized in the right lower abdomen after three or four days. There was a past history of two previous attacks of abdominal pain diagnosed as appendicitis.

The patient was acutely ill, poorly nourished and had a fever of 104°F. There was muscle spasm, acute tenderness and a 6 cm. mass in the right lower abdomen. The liver area was tender and the superficial veins of the abdominal wall were dilated. The white blood count was 17,000, cephalin flocculation 4+, thymol turbidity 2.6, icterus index 19, prothrombin time twenty-four seconds with a control of fifteen seconds. X-ray examination of the abdomen showed free air in the right flank, stepladder distention of the small bowel and a coprolith in the right lower abdomen. Blood culture was normal. The patient ran a septic fever to 105°F. daily. He was treated with a Miller-Abbott tube, large doses of penicillin and streptomycin, 250 cc. of whole blood daily and intramuscular heparin for the first two weeks of his hospital stay. An abscess in the right lower abdomen was drained on the seventh day and the pus cultured Esch. coli. The patient's fever subsided. After two cephalin flocculation determinations of 4+, a negative was obtained a month after admission. The icterus index dropped to 8 and bromsulfalein excretion was normal. The patient was discharged on his twenty-ninth hospital day. The appendix was removed four months later and showed healing appendicitis and periappendicitis. Liver biopsy was normal.

COMMENT

The incidence of pylephlebitis and liver abscess has decreased in recent years due to earlier diagnosis and treatment of appendicitis and possibly somewhat to the rather general use of antibiotics. The history of appendicitis, however, may be atypical and confusing as in most of the cases in the present series, accounting for a delay in diagnosis. The chill or better multiple chills as pointed out by Colp[3,4] are helpful when present. The presence of a laminated coprolith or fecalith in the right lower quadrant on a flat roentgenogram of the abdomen serves to focus attention on the appendix as a possible source of inflammation. There were three coproliths in the six recent cases in this series. In a group of 100 acutely inflamed appendices removed consecutively, Remington[16] reported that fecaliths associated with gangrene and rupture of the appendix were more frequent in the twenty-six cases showing thrombosis of the appendiceal veins.

Once a diagnosis of pylephlebitis is suspected, vigorous supportive therapy is necessary in view of the prolonged and wasting nature of this process. Multiple transfusions help combat the anemia and hypoproteinemia which develop. Antibiotics can sterilize the blood stream as evidenced by blood cultures becoming negative, and every effort should be made to choose the proper drug by sensitivity tests when the causative organism can be cultured. Anticoagulant therapy probably is useful judging by a very limited experience with it. It may be able to prevent the spread of the thrombosis to the larger veins while the antibiotics deal with the organisms in the blood stream. Heparin in 50 mg. doses is probably preferable to dicumarol in view of the severe toxic depression of the liver which is present.

In spite of all the therapeutic methods available, the mortality of pylephlebitis with liver abscess remains high. The prognosis with a single liver abscess is better than with multiple abscesses as shown by Ochsner[13] who found a mortality of 37.5 per cent with a single compared to 95 per cent for multiple abscesses. Eliason[6] was of the opinion that a single abscess was due to a septic embolus from a circumscribed venous infection and had a fairly good prognosis following surgical drainage. Of his fourteen cases of pylephlebitis with liver abscess, seven had single abscesses and lived. Two of the three survivors in the present series had single large abscesses in the right lobe of the liver though one was multiloculated. It remains to be shown whether specific antibiotic and anticoagulant therapy can

overcome pylephlebitis with multiple small liver abscesses.

SUMMARY

1. Ten of seventeen cases of pylephlebitis with liver abscess were due to acute gangrenous or perforated appendicitis, an incidence of 59 per cent. The other seven cases were due to other suppurative disease in the pleural or peritoneal cavities.

2. Diagnosis was difficult to make clinically due to the atypical history and findings. Shaking chills before or shortly after the onset of symptoms and a coprolith in the right lower abdomen by x-ray examination were helpful when they were present.

3. Jaundice was an important manifestation of the condition being present in twelve of seventeen cases with icterus indices generally from 19 to 25.

4. Blood cultures were positive in nine of the thirteen patients tested with the predominant organism being Esch. coli followed by hemolytic streptococcus and hemolytic staphlococcus aureus either separately or associated with coli-aerogenes organisms.

5. Treatment since 1946 consisted of drainage of appendiceal, subphrenic or liver abscesses where indicated, antibiotics chosen by sensitivity test when the organism was available for culture, anticoagulant therapy with heparin and supportive measures such as frequent blood transfusions.

6. Mortality in the eleven cases prior to 1946 was 100 per cent while it was 50 per cent in the six cases since that time. Two of the three patients who survived had drainage of single abscesses in the right lobe of the liver.

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Observations on Postoperative Pancreatitis and Postoperative Elevation of the Serum Amylase

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ACUTE pancreatitis has been recognized for many years by the medical profession as a complication that may follow various operative procedures. Schmieden and Sebening[1] in 1928 emphasized the relationship of surgery of the pancreas and neighboring organs in the occurrence of postoperative pancreatitis, and numerous other authors[2-8] have added case reports and other contributions since that time. More recently Millbourn,[9] Metheny and Lundmark,[10] Warren[11,12] and Dunphy[13] have re-emphasized the occurrence of this complication.

There is little exact knowledge concerning the etiology of acute pancreatitis, either as a primary disease or as a postoperative complication. Experimental and clinical observations suggest a number of different factors that may play a role, among which are: (1) obstruction at sphincter of Oddi, with reflux of bile up the pancreatic duct and activation of pancreatic enzymes—the "common channel" theory; (2) obstruction of the pancreatic duct (by disease or spasm) and simultaneous stimulation of production of external pancreatic secretion; (3) impairment of a portion of the vascular supply of the pancreas by injury or disease; (4) direct injury of the pancreatic ducts; (5) infection.

It is fortunate that the fulminating type of postoperative pancreatitis is rare, since it strikes with the unpredictability and fury of a lightning bolt. Milder forms of the disease, if they exist, are probably masked by the diffuse discomfort of an abdominal operation. It occurred to the writers that a routine study of the level of the serum amylase in the immediate postoperative period might offer a clue to the occurrence of mild pancreatitis.

Eighty-five cases were available to this study, unselected except for the facts that all were patients of one of us (S. O. H.), and all patients had operations of the upper abdomen. Determinations of the serum amylase were made once before operation and three times afterward, if possible on the first, second and third postoperative days. The clinical laboratory made the determinations, employing the method of Myers and Killian, in which the upper limit of normal serum amylase is taken to be 200 units. A level of 500 units or greater was arbitrarily selected as representing a significant elevation. (If 300 units had been selected, sixty-five of the eighty-five patients

TABLE 1

POSTOPERATIVE ELEVATION OF SERUM AMYLASE (500 UNITS OR MORE) IN RELATION TO MANIPULATION OF THE PANCREAS

	No. of Patients	No. of Patients with Postoperative Elevation of Serum Amylase
Direct manipulation of pancreas:		
Surgery involving pancreas (including gastric resection, etc., see text)	19	9
Common duct exploration	23	8
Cholangiography without exploration	6	0
Total	48	17
No direct manipulation of pancreas	37	10
Grand total	85	27

would have had at least one higher postoperative level.)

Seventy-four of the eighty-five patients each had an operation for primary disease of the stomach, duodenum, gallbladder or bile ducts. Six had primary disease of the pancreas (although not necessarily an operation directly upon the pancreas); two had cirrhosis of the liver; two had diseases of the spleen; and one had hepatitis. Nearly one-third[27] of these patients had an elevation of serum amylase to 500 units or more for at least one postoperative determination. Attempts were made to correlate these "significant" elevations with manipulation of the pancreas at the time of operation, with pre-existing disease of the pancreas, and with the type of operative procedure.

TABLE II
POSTOPERATIVE ELEVATION OF SERUM AMYLASE (500 UNITS OR MORE) IN RELATION TO PRE-EXISTING PANCREATIC DISEASE

Clinical Status of Pancreas	No. of Patients	No. of Patients with Postoperative Elevation of Serum Amylase
Normal pancreas	55	15
Primary disease of pancreas	15	3
Secondary involvement of pancreas by extension (ulcer or cancer)	15	9
Total	85	27

Table I compares postoperative levels of serum amylase in patients having had direct manipulation of the pancreas (such as would occur with a partial resection of the organ, a biopsy or a gastric resection in which ulcer or malignant disease involved the pancreas), and patients having had no direct manipulation of the pancreas. The incidence of serum amylase elevation in the one category is not notably different from that in the other. Table II compares the postoperative levels of serum amylase in patients with and without pre-existing pancreatic disease. Again, there is no evident correlation.

The incidence of increases in the postoperative levels of serum amylase in relation to various operative procedures is analyzed in Table III. Gastric resection and exploration of the common bile duct are shown to be followed by a high incidence of such elevations. In fact, if the series is arbitrarily divided into two groups (Table IV), the first including only gastric resection, common duct instrumentation and operations upon the pancreas, and the second including all of the other operative procedures (including procedures such as vagotomy and gastroenterostomy, simple cholecystectomy, surgery of the spleen), there is a statistically significant increase in incidence in the first group over that in the second. This suggests that the operative procedure *per se* plays a greater role than that of the other factors considered.

TABLE III
POSTOPERATIVE ELEVATION OF SERUM AMYLASE (500 UNITS OR MORE) IN RELATION TO TYPE OF OPERATIVE PROCEDURE

Type of Procedure	No. of Patients	No. of Patients with Postoperative Elevation of Serum Amylase
Gastric resection	35	16
Vagotomy and posterior gastrojejunostomy	8	2
Exploration of common bile duct	23	8
Operations upon pancreas (for primary pancreatic disease)	3	1
Simple cholecystectomy	6	0
Miscellaneous	10	0
Total	85	27

There were two fatal cases of pancreatitis in this series.*

CASE REPORTS

CASE I. A sixty-six year old white man, a diabetic, complained of intermittent postprandial and nocturnal epigastric discomfort

* Fatalities from pancreatitis are not as common as the occurrence of two in this small series of eighty-five cases might imply. Actually, Case I is the only fatality from pancreatitis (or any other cause) in nearly 300 elective or semi-elective operations for benign gastric, duodenal or jejunal ulcer performed from January 1, 1950, through March, 1954. Similarly, Case II is the only fatality from pancreatitis in more than 300 operations for gallstones performed in the same period of time.

of three years' duration. The patient had lost 15 pounds in weight. Preoperative studies demonstrated cholecystolithiasis, a large gastric ulcer suspected of being malignant, diabetes and impaired liver function attributed to cirrhosis of the liver. At operation, a subtotal gastric resection (anterior Polya) was performed for a large, benign gastric ulcer penetrating deeply into the pancreas. No effort was made to excise the surface of the ulcer by cutting into the pancreas, and all manipulation was directed only toward freeing the stomach from the pancreas. A cholecystectomy and a feeding jejunostomy were also performed. On the first postoperative day there was profuse drainage of bile and pancreatic secretion through the stab-wound drain. The serum amylase level was 277 units before operation; on successive determinations it was, respectively, 1,433, 1,619, 58, 69 and 287 units. In spite of vigorous support, the patient died twelve days after operation. Postmortem study showed an acute suppurative and necrotizing pancreatitis, generalized peritonitis and Laennec's cirrhosis of the liver. The suture lines were all intact.

TABLE IV

POSTOPERATIVE ELEVATION OF SERUM AMYLASE (500 UNITS OR MORE) IN RELATION TO ARBITRARILY GROUPED TYPES OF OPERATIVE PROCEDURES

Group	No. of Patients	No. of Patients with Postoperative Elevation of Serum Amylase
Gastric resection	35	16
Exploration of common bile duct	23	8
Operations upon pancreas (for primary pancreatic disease)	3	1
Total	61	25 (41%)*
Vagotomy and posterior gastro jejunostomy, simple cholescystectomy, etc.	24	2† (9%)*

* The difference is statistically significant.

† Both cases in vagotomy and posterior gastrojejunostomy.

Comment. The manipulation involved in freeing the stomach from the pancreas was evidently sufficient to spread infection in an already diseased organ in a patient who was elderly and debilitated by diabetes and cirrhosis of the liver in addition to his surgical diseases.

CASE 11. A thirty-eight year old white woman complained of jaundice of six weeks' duration, which had followed two weeks after the birth of a normal baby, her ninth pregnancy. There had been colicky pain, light stools and dark urine. She had lost 45 pounds in weight. Laboratory studies were consistent with a diagnosis of obstructive jaundice. At operation many stones were found in the common duct. Cholecystectomy and choledocholithotomy were carried out easily, the only unusual feature being the reopening of the common duct for extraction of an overlooked stone that was demonstrated by the completion cholangiogram. On the first day after operation tachycardia with hypotension developed, although there were no outstanding abdominal findings. The patient continued to exhibit evidences of collapse and was re-explored on the second day. (A serum amylase determination was 1,757 units and subsequent values were 1,705 and 490 units.) A hemorrhagic pancreatitis was found and the pancreas drained, but the patient died two days later in spite of every effort to save her. Postmortem examination confirmed the existence of extensive acute hemorrhagic pancreatitis with peritonitis.

Comment. Although the operative procedure was technically easy and no different in any obvious way from that performed on many other patients whose convalescences were uneventful, it must be assumed that the operative manipulation precipitated, in some undetermined way, the fatal complication.

OBSERVATIONS

The unexpected finding of this study is the high incidence of serum amylase elevation during the postoperative course of the patients in this series. Since there were very few instances in which clinical evidence suggested acute pancreatitis (except for the two fatal cases cited), one may conclude that minor derangements of the pancreas are relatively common after surgery involving the pancreas directly or indirectly. It is not possible to prove that the elevated serum amylase does, in fact, reflect a mild pancreatitis in every instance, but it suggests that such an interpre-

tation be seriously considered. If one accepts this view, the clinician will be well advised to consider the possibility of a mild acute pancreatitis in patients who have had operations on the pancreas, or in its vicinity, when the postoperative course is not so smooth as had been anticipated.

SUMMARY

A study of the levels of the serum amylase during the first few days after surgery of the upper abdomen in a series of eighty-five unselected patients reveals that in twenty-seven (32 per cent) there was an elevation of 500 units or greater on one or more determinations. (Two hundred units is the accepted upper limit of normal for the technic employed.) By chance, there were two fatal cases of pancreatitis in this group, but very few additional patients had clinical findings suggestive of acute pancreatitis, irrespective of the elevated levels of serum amylase. There is no proof that the postoperative elevations of serum amylase in these patients actually reflect derangement of the pancreas, but such elevations suggest that numerous mild cases may pass unnoticed. There was no apparent relation between postoperative increase in the serum amylase level and pre-existing pancreatic disease, or to direct surgery upon the pancreas, but it was noted that gastric resection and instrumentation of the common duct were followed by an increased incidence of such elevations. It is suggested that pancreatitis be considered in the differential diagnosis of the milder disorders following in the first few days after operations on stomach, duodenum, bile ducts and pancreas.

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Use of the Knott Technic of Blood Irradiation Therapy in Cases of Threatened and Inevitable Abortion

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THE prevention of spontaneous abortion is a recurrent problem faced by the obstetrician and general practitioner alike. It is not merely one of wastage of fetal life early in pregnancy. Investigation by many workers in the field reveals this abortion-to-stillbirth ratio; the frequency of stillbirth is greater in those women who have previously suffered from abortion than those who have not. In addition, many patients who experience stillbirth have threatened to miscarry during that particular pregnancy. There is also a high incidence of twins and fetal malformations in groups showing abortion sequences.[1]

It should be pointed out that the cases presented in this report include inevitable or incomplete abortions, as well as threatened abortions. The problems of infection and prolonged morbidity in these cases of incomplete abortion are all too well known to the surgeon and obstetrician and require no comment at this time.

The cases set forth herein are not presented as a complete study, but rather to stimulate further evaluation of the use of blood irradiation therapy in threatened and inevitable abortions. However, while the series reported is relatively small, the findings are believed to be significant.

Incidence of Abortion. Available figures on the incidence of spontaneous abortion indicate that between 10 and 20 per cent of all pregnancies end in abortions. Eastman,[2] after a study of 100,000 pregnancies, concludes that 10 per cent may be expected to abort, and Williams[3] found that in 20 per cent of all pregnancies there was miscarriage.

Malpas[4] concludes from analyses of his own cases and those of the late Sir Beckwith Whitehouse that spontaneous abortions can be classified as follows: (1) those in which the abortion resulted from a random or fortuitous factor not likely to be repeated, and (2) those in which there is a permanent or recurrent factor. Malpas' analysis presents the conclusion that only about 1 per cent of all pregnant women abort because of recurrent factors and that a recurrent factor may be inferred with reasonable certainty after the recurrence of three successive abortions.

Etiology. Brown[1] points out that it is highly probable that the uterus never expels a healthy embryo, and that in every case of abortion the fetus is either dead or dying. Although the intrauterine environment is very safe, embryonic variations do occur. Such conditions are either of genetic origin or due to the reaction of the growing embryo to an abnormal intrauterine environment.

He also observes the altered growth or death of the embryo occurring when the composition of the mother's blood is unfavorable as, for example, in diabetes, nephritis or syphilis or when the mother suffers from a disease or malnutrition. He states that virus disease can be transmitted through the placenta to the embryo and cites as an example the case of a mother suffering from smallpox who may either kill her embryo or produce a pock-marked child.

Recently the discovery of polio virus in a three month old fetus was regarded as a most important step forward in polio research.

Summarizing, Brown comments that primarily "abortions, abnormalities and fetal deaths are due to the influence of illness or disease of the mother upon the developing fetus."

Diagnosis. The early symptomatic diagnosis of threatened abortion is always open to question. Vaginal bleeding may result from relatively innocuous causes.[5] However, from a practical viewpoint the following symptoms may be considered as an early sign of threatened expulsion of the fetus, unless there is some

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other demonstrable cause: (1) all instances of vaginal bleeding and/or low abdominal cramps and (2) premature labor. It was on the basis of these criteria that the thirty patients presented in this series were regarded as threatening to abort.

CONSIDERATION OF BLOOD IRRADIATION THERAPY (KNOTT TECHNIC)

The Knott technic of blood irradiation consists of withdrawing and citrating 1½ cc. of the patient's blood for each pound of the patient's body weight. This blood is then passed through the Knott Hemo-Irradiator which accurately regulates the return flow through a chamber with a quartz glass window. There the blood is exposed to ultraviolet rays at close range and is returned to the patient by the same needle through which it was withdrawn.

In 1947 Olney[6] reported five cases of threatened abortion in patients with pelvic cellulitis treated successfully with the Knott technic of blood irradiation therapy, and commented as follows: "The abortion had progressed to a point of regular, frequent pains, heavy bleeding, and seemed inevitable in each case. All these symptoms subsided promptly with ultraviolet blood irradiation treatment, and the patients continued to full term and normal delivery."

Rebbeck in 1942[7] and again in 1951[8] reported excellent results obtained through the use of the Knott technic of blood irradiation therapy in cases of postabortional sepsis and commented: "Definite protection was afforded against the usual disastrous spread of uterine infection in the pelvis or into the blood stream. In cases of pronounced sepsis a remarkable reduction in the manifestations of toxemia was achieved."

Toxemia of pregnancy and pelvic inflammatory processes are among the chief causes of abortion. With these considerations in mind, and being cognizant of the fact that the uterus rarely expels a healthy embryo, it was believed that the following demonstrable benefits of application of the Knott technic of blood irradiation as reported in the literature appeared to offer assistance in the prevention of threatened abortion and the avoidance of complications in the incomplete abortion: (1) Decrease of edema and inflammatory processes;[6,11] (2) decrease of toxic symptoms;[7,12] (3) increase of venous oxygen[9] and (4) increased phagocytosis.[10]

Conventional therapies seemed to have little to offer in cases of threatened abortion. Colvin et al.[13] comment as follows: "At the present time, therefore, the usual treatment of threatened abortion is a combination of bed rest, sedation, endocrine and vitamin therapy. The procedure is expensive, incapacitating to the woman, and the results, as a rule, reveal the futility of such a plan of management." Therefore blood irradiation therapy was administered to thirty patients during thirty-eight episodes of vaginal bleeding, low abdominal cramps (or both) and premature labors. No other treatment was given except as noted in the tabulation of cases presented herein.

CASE REPORTS

CASE 6. This woman was one of the first gravid patients to receive blood irradiation therapy. Her two previous pregnancies had terminated with 3¾ and 3½ pound incubator babies, respectively. When, during her third pregnancy, she began having uterine contractions at the seventh month, it appeared her pregnancy would culminate in the same manner as her two previous ones. One treatment of blood irradiation was administered and the patient's cramps ceased promptly. Pregnancy continued uneventfully to the thirty-third week when delivery was induced due to generalized edema which had appeared three weeks previously despite low salt diet. The patient was delivered of a normal, healthy 6 pound baby.

CASE 8. This patient was given blood irradiation treatment prophylactically for the first time in the fifth month of her third pregnancy. Her first pregnancy had terminated at seven months with a stillbirth and her second resulted in a miscarriage at three and a half months. When she first presented herself to us for examination at five months, she expressed concern as to the outcome of that pregnancy because of her two previous experiences. She was given one treatment of blood irradiation. Her pregnancy continued uneventfully to normal delivery at term.

CASE 25. This patient had had one miscarriage and had been delivered of two babies who survived only a few hours after birth. The patient had a sudden onset of brisk vaginal bleeding and low abdominal cramps at three and a half months. Three hours after the appearance of these symptoms blood irradia-

TABLE 1
SUMMARY OF DATA

Case No.	Pregnancies (No.)	Duration of Pregnancy (Mo.)	Symptoms	UBI* Administration (Date)	Results Observed Following Treatment
1	3	4	Cramps 12 hr.	5/7/51	Cramps ceased; delivered at term
2	2	3	Flow 4 hr.	1/17/51	Flow ceased; delivered at term
3	4	4½	Cramps, spotting	8/10/49	Term delivery; Bandl's ring; retained placenta; breech
4†	2	3	Flow 2 days	7/31/49	Flow ceased; passed placenta 2 days later; no fetus found
5	2	6	Cramps, flow	1/11/49	Symptoms ceased; delivered 3 wk. prior to due date
6	3	7	Cramps	9/20/48	Cramps ceased; delivered 6 lb. baby at 33rd week (2 previous pregnancies resulted in 3¾ lb. and 3½ lb. babies, respectively; all lived)
7	2	2	Cramps 3 hr.	11/8/50	Cramps ceased soon after UBI
		3½	None	12/23/50	Treatment given prophylactically
		7½	Intermittent cramps, 3 days	5/5/51	Less cramps after UBI; flow began 5/20/51; bed rest 6 days; delivered at term satisfactorily
8‡	3	5	None	10/12/49	Previous miscarriages at 7 and 3½ mo., respectively; UBI administered prophylactically; normal delivery at term
9†	1	2	Cramps, spotting	9/11/51	Placental tissue passed next day; no fetus found
10†	3	3	Cramps, spotting 12 hr.	12/11/50	Next day passed clots and macerated placenta
11	4	7	Cramps	9/25/50	Cramps ceased; delivered at term
12†	4	2½	Flow, cramps 12 hr.	11/11/51	Had passed several large clots before UBI; fetus not found; flow subsided gradually
13	1	7½	Cramps 4–6 min.	12/1/51	Cramps ceased; delivered at term
14†	4	2½	Flow 12 hr.	7/6/51	Passed large clots; no fetus found after UBI
15†	2	5	Flow 3 wk.	1/22/51	Delivered dead fetus 4 hr. after UBI; half of placenta showed old separation
16†	1	3	Cramps, spotting 1 day	6/2/49	Passed 2 cm. dead fetus intact in sac 8 hr. after UBI
17	2	3½	Intermittent cramps 2 days	12/28/51	Cramps ceased
		7	Cramps 5 min.	4/2/52	Membrane had ruptured 6 hr. before UBI; 3 lb. fetus lived 4 hr., died of atelectasis; marginal placenta previa
18†	2	5½	Cramps	9/11/51	Expelled 4 mo. size macerated fetus 6 hr. after UBI
19	2	4	Cramps, flow	5/11/50	Flow decreased after UBI; bed rest 2 days
			Spotting	5/15/50	Symptoms ceased
		5	Cramps	6/18/50	Cramps ceased
		8	Cramps, spotting	9/19/50	Symptoms ceased; delivery by cesarean section 10/24/50 a/c previous section
20	1	4	Spotting and cramps	9/20/48	Symptoms ceased; delivered at term; low forceps
21	1	5½	Cramps and spotting	5/13/49	Symptoms ceased; delivered at term
22‡	4	2½	Cramps, flow 1 wk.	11/27/49	One previous miscarriage; flow and cramps stopped after UBI
23	1	7	Intermittent cramps	9/11/51	Cramps ceased; delivery at term
24	1	3	Cramps, spotting	1/15/51	Symptoms ceased; delivery at term
		3	Flow 3 hr.	5/8/52	Flow ceased; due 10/17/52
25‡	6	3½	Flow, cramps	9/20/51	Symptoms ceased; delivered at term (only survival of last 4 pregnancies)
26	3	2½	Flow	2/28/51	Flow ceased; delivered at term
27	8	6	Flow	7/28/51	Flow ceased; term delivery
28	4	7	Flow	1/5/52	Flow ceased; term delivery
29	1	3	Flow	3/9/51	Flow ceased; term delivery
30	2	3	Flow	9/4/48	Flow decreased after UBI
			Spotting yet	9/20/48	Spotting ceased
		5	Spotting 1 day	11/20/48	Spotting ceased; term delivery
	3	2	Cramps, spotting	9/20/50	Symptoms ceased; delivery 1 mo. premature; 4½ lb. baby lived

* Ultraviolet blood irradiation.
† Incomplete or inevitable abortion.
‡ History of previous abortions.

tion therapy was administered. Her symptoms ceased approximately two hours after the treatment and her pregnancy continued uneventfully to normal termination.

Table 1 lists all patients and episodes included in this series.

SUMMARY

Thirty consecutive patients with thirty-eight episodes of low abdominal cramps and/or vaginal bleeding and/or premature labors occurring during their pregnancies were treated with the Knott technic of blood irradiation. It was believed that these symptoms indicated the probability of threatened or inevitable abortions.

Eight of these patients had incomplete abortions. Three passed dead fetuses after administration of blood irradiation treatment; five passed only placentas after treatment. Curettage was not necessary in any of these eight cases. The uterus returned to normal promptly, infection did not appear and the patients recovered rapidly.

Twenty-one patients with thirty episodes of low abdominal cramps and/or vaginal bleeding and/or premature labors were treated with the Knott technic of blood irradiation, usually within twenty-four hours after the appearance of the symptoms (in two cases the treatment was not administered until forty-eight and seventy-two hours after the onset of the symptoms, respectively). In each case the symptoms ceased promptly and all patients in this group continued to term or near-term and were delivered of normal babies.

One patient (Case 8) was given blood irradiation treatment as a prophylactic measure during her third pregnancy due to a history of two previous pregnancies, one of which terminated in a stillbirth at seven months and the other in a miscarriage at three and a half months. The patient's pregnancy proceeded uneventfully to normal delivery at term.

In only one patient (Case 17) was the treatment unsuccessful. This pregnancy ran a rather stormy course due to marginal placenta previa.

Three patients had histories of previous abortions. One patient (Case 8) reported two previous miscarriages at seven and three months respectively, another (Case 22) presented a history of one previous miscarriage and another (Case 25) reported three previous miscarriages.

It is interesting to note that rapid subsidence of cramps and bleeding was observed after administration of blood irradiation therapy in each of the thirty episodes previously mentioned, as well as obviation of the necessity for curettage and prompt recovery of the patient in the cases of inevitable or incomplete abortions.

CONCLUSIONS

The rate of spontaneous recovery in patients not having a history of previous abortions is reported as being from 70 per cent[13] to 90 per cent.[2] Although the series presented herein is small, it is interesting to note only one failure in twenty-one patients, representing a salvage rate of 95.2 per cent. Thus the rapid subsidence of cramps and/or bleeding and/or premature labors noted in the aforementioned patients after the administration of blood irradiation therapy is significant.

Clinical observation of this series indicates that the Knott technic of blood irradiation therapy is a valuable adjunct to the treatment of threatened abortion and in the prevention of immediate and delayed complications in inevitable abortion.

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Surgical Treatment of the Postphlebitic Syndrome

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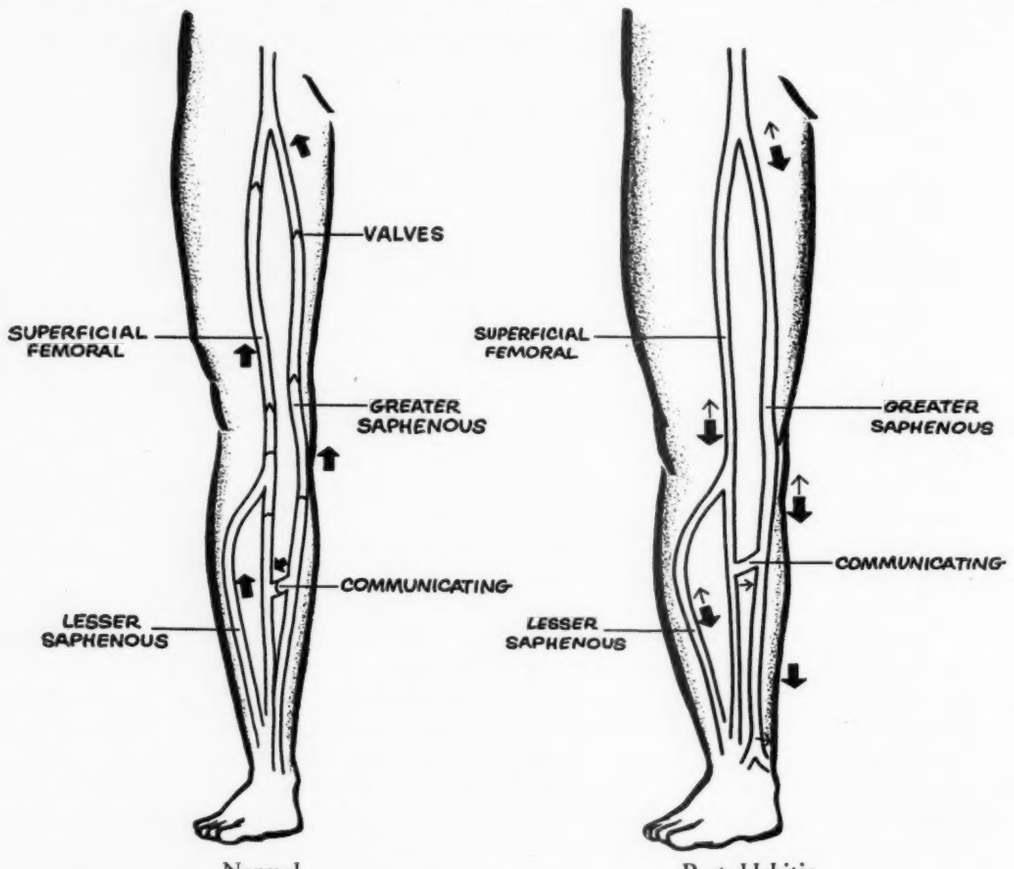
"MILK LEG" has always been a dreaded complication of any medical, surgical or obstetric situation. Until the early 1940's the accepted mode of treatment was complete bedrest from four to six weeks with the leg elevated and covered with ice bags. Bedrest was deemed necessary in order that the thrombus in the vein would not detach itself and be carried to the lung. Ice was thought to be advantageous because it cut down the blood supply to that part and organization of the clot would be hurried. Acute thrombophlebitis or phlegmasia alba dolens was not differentiated from phlebothrombosis which has no infected clot but one which is friable, easily detached and often silent. After the acute phase of thrombophlebitis had subsided, the patient was doomed to a swollen leg which usually resulted in a chronic ulcer or recurrent attacks of erysipelas. It was then believed that all that could be done for such a condition was to apply an elastic stocking or bandage and treat the ulcer itself. In time high ligation of the saphenous vein was thought to be a help; many was the skin graft laid on the denuded surface which appeared healthy until the leg became dependent and then the graft was seen to melt away.

Naturally the best treatment for the postphlebitis syndrome is prevention. Early ambulation, adequate hydration, better anesthesia and improved surgical technic have all contributed to a decrease in this complication. Early ambulation was thought to be the answer, but in the larger medical centers this is found disappointing in its results. Much progress has been made in the treatment of acute thrombophlebitis. In 1934 Leriche[2] and his co-workers established the efficacy of repeated lumbar sympathetic block to relieve the concomitant vasospasm, reduction in swelling, rise of temperature and other symptoms. Anticoagulant therapy has been of further help. However, the understanding of the thrombophlebitic clot as compared to the phlebothrombotic clot has given us courage to bind these legs in elastic bandages and have the patients ambulatory.

In acute thrombophlebitis an infected thrombus will be found in the superficial femoral, which vessel is a continuation of the posterior tibial by way of the popliteal. Furthermore, an infected thrombus in the vast majority of cases is also present in the greater and lesser saphenous veins and their branches. For years we were threatened with dire consequences if operative interference of the greater and lesser saphenous were undertaken in a postphlebitic situation. It was believed that the clot which had organized had become fibrous and attached to the intima of the vein and so occluded these veins that further interruption of return blood flow would result in necrosis of the extremity. Buxton[1] and Linton,[4] however, have shown the fallacy of this view. They demonstrated that the thrombus in the superficial femoral recanalized and that the hydrostatic pressure of the blood in an incompetent deep vein is the major cause of the typical postphlebitic syndrome. Clinically we see a leg which is edematous with brawny induration, especially on the inner aspect, and more often than not an ulcer just above the internal malleolus. Furthermore, there will be varicosities of both greater and lesser saphenous systems and occasionally ulcers posterior to the external malleolus. This clinical picture is easily understood when we see the pathologic changes, for in this situation there is inadequate return of blood flow with resulting edema, piling up of metabolites and the destruction of the barriers of repair due to the inadequacy of the valves in these veins. (Fig. 1.) Linton[4] has performed many thousands of superficial femoral ligations and has seen those patients in whom the vein was not interrupted to give the aforementioned syndrome. He also states that after three

unsuccessful skin grafts in five years on one woman he ligated and divided the superficial femoral in desperation, with an excellent result and "the lady could take her first bath in seventeen years."

Shumaker[5] and others have shown the hydrostatic pressure in the posterior tibial vein on walking to be greater after ligation of the superficial femoral than before. However, "the proof of the pudding is in the eating" and the good results now seen following surgical intervention more than outweigh the theoretic deductions obtained by some. From a systemic standpoint an occasional patient will present an overstimulation of the sympathetic with concomitant vasospasm of that extremity. This is not entirely understood but is a factor that must not be forgotten.



Normal Postphlebitic

FIG. 1. Diagrammatic sketch of the larger veins in normal and postphlebitic situation.

Treatment begins with healing the ulcer. Bedrest, hot applications and elevation of the affected extremity will cause any of these ulcers to heal, unless they have been of such long-standing that scarification prevents epithelialization. This mode of therapy is expensive and economically unnecessary. One to three days in the hospital to allow swelling to subside and to rid the ulcer of secondary infection is sufficient. Some type of compression dressing to divert the returning blood flow to the adequate venous system will cause the ulcer to heal in several weeks. I have found nothing superior to the old-fashioned Unna boot which is so easily applied and which gives such effective and even compression over the entire lower extremity. This is applied before the patient is allowed out of bed and is changed every week or two until the ulcer has healed or until healing has become stationary. The patient is then readmitted to the hospital to effect operatively what the Unna boot has done temporarily. The operation consists of: (1) Ligation of the superficial femoral and division of this vein where it joins the profunda; (2) high ligation of all branches of the greater saphenous at the saphenofemoral junction, and stripping of the greater saphenous from the

groin to the internal malleolus and (3) ligation of the lesser saphenous at the popliteal line and stripping of this vein from that site to posterior to the external malleolus. Elastoplast is applied from behind the toes to the groin and ambulation begun the afternoon of the operation. This elastoplast is kept on for two weeks. After that the patient is instructed to wear either an elastic stocking or ace bandage for from four to six months. This is absolutely necessary so that swelling will not be excessive until a good collateral circulation is established in the lower portion of the lower leg. The veins through the muscles and especially in the inner aspect of the lower leg must be given time to enlarge for an adequate return of venous flow to be accomplished. Whenever the ulcer has not been healed by boots, a split-thickness skin graft is carried out at the time of the aforementioned procedure. If after a reasonable length of time swelling is still excessive, lumbar sympathetic block is performed to see if there is overstimulation of this nervous system. If two lumbar sympathetic blocks help the patient, lumbar sympathectomy of the affected side is advocated.

TABLE I

Procedure	Cases	Recurrence of Ulcer	Recurrence (%)
Ligation of saphenous (1942–1948)	8	8	100
Ligation and stripping (1948–1950)	7	6	85
Ligation of superficial femoral and stripping (1950–1952)	20	0	0

The criterion of a good result, therefore, is not only a continued healed ulcer site but also that swelling and discomfort will be minimal. This series is extremely small to speak of in terms of percentages but the statistician demands subjugation to figures.

Table I lists three types of procedures and their results. No case has been included beyond September, 1952, because of the length of time involved in obtaining an adequate return blood flow and evaluating the efficacy of this procedure. It is of interest that some of the strippings were done on the patients who suffered a high ligation first between the years of 1942 to 1948. Two of the six with ligation and stripping had only the superficial femoral explored and surrounding scar excised.

Table II shows that 1 of 5 undergoing this procedure still has attacks of recurrent erysipelas. I would like to mention here that segmental thrombophlebitis seen in generalized varicose veins is not the situation listed under erysipelas.

TABLE II

Procedure	Cases	Recurrence of Erysipelas	Recurrence (%)
Ligation of saphenous (1942–1948)	3	3	100
Ligation and stripping (1948–1950)	3	3	100
Ligation of superficial femoral and stripping (1950–1952)	5	1	20

TABLE III

Procedure	Cases	Recurrence of Swelling	Recurrence (%)
Ligation of saphenous (1942–1948)	8	8	100
Ligation and stripping (1948–1950)	7	7	100
Ligation of superficial femoral and stripping (1950–1952)	20	2*	10

*Lumbar sympathectomy with complete relief.

Table III shows results in regard to post-treatment swelling. Linton[4] reported a further refinement of this technic in which he excised part of the deep fascia on the internal aspect of the lower leg. He does this because he believes that lymphedema is due to the scarification of this fascia and a good result cannot be obtained unless this refinement is added. Of the two individuals who had a recurrent swelling one has undergone a lumbar sympathectomy with complete relief while the other refuses to undergo the tests or operation.

SUMMARY

We find that treatment of acute thrombophlebitis has changed radically in the last

decade. It is hoped that more adequate therapy based on better understanding of these pathophysiologic changes will reduce the number of patients suffering from a postphlebitic syndrome. Present day treatment of the postphlebitic syndrome, once it has become chronic and disabling, is encouraging both to the patient and to the physician.

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Patey and Nurick treat cystic disease of the breast by simple aspiration. They suspect an intracystic growth (requiring surgery) if the aspirated fluid is bloody. If the lump persists after aspiration or if the cyst rapidly refills within a few weeks, they also believe biopsy is indicated. Of course the aspirated fluid should always be examined for malignant cells. (*Richard A. Leonardo, M.D.*)

The Treatment of Keloids with a Combination of Hyaluronidase and Kutapressin

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THE solution of the perfect or near perfect management of the keloid problem has for years intrigued the members of the medical profession generally, and especially those specializing in surgery, radiology and dermatology.

Not infrequently the latter three specialties, through their combined or pooled efforts and procedures, have produced the best results and contributed the brightest rays of hope for ultimate success. These conclusions have been drawn after a number of years of careful study of several hundred keloids and hypertrophic scars. During this time the author tried and used many methods, both singly and in several combinations. These varied from gentle massage with bland greases through the gamut of tissue drainage, chemical infiltrations, electrosurgical procedures, freezing with carbon dioxide snow and surgical excision. We are now convinced that we can safely recommend a most effective and simple procedure for use in treatment of keloids and hypertrophic scars. This method may, in some cases, be all that is necessary for obtaining excellent results.

The procedure almost always is an excellent preparatory measure when surgery and physiochemical methods are to follow. Doubtless the most satisfactory single method for the treatment of keloids and hypertrophic scars has been by the use of radiotherapy.

Surgical removal either by scalpel or the electrocutting current have too frequently been followed by further cicatrization. The use of solid carbon dioxide has given fair results in some cases.

Cornbleet's work on the treatment of keloids with the use of hyaluronidase injected into the lesions, thereby producing softness and at times resolution, has given much hope for the management of keloids.

Marshall and Schadeberg report favorable results in the management of keloids and related conditions with the use of kutapressin® (a fractionated preparation derived from crude liver).

Nierman, and Pensky and Goldberg have reported studies in the management of acne vulgaris (especially the cystic and refractory types) by using kutapressin, and those workers stress the importance of the vasoconstrictor properties of this preparation.

This is a preliminary report on our use of hyaluronidase and kutapressin, both singly and in combination, in attempts to reduce the size of and to soften both hypertrophic scars and keloids. When either preparation was used alone, we had fair to moderate success in achieving our goal. More recently we have been using these two preparations in combination and our results have been most gratifying. A few of these combined treated cases have been followed with roentgen therapy or solid carbon dioxide therapy, and one case with both of the latter methods. To date none have been followed with surgery. Our preliminary studies have convinced us from the basically striking results that the use of kutapressin and hyaluronidase in combination is probably the best method for treating keloids and hypertrophic scars with the aim of causing softening and resolution.

When physical, chemical and/or surgical methods are to be used in the management of keloids and related conditions, excellent results can be expected if the lesions are previously treated by the injection of a combination of hyaluronidase and kutapressin.

To date we have treated and have under observation twenty-seven patients, as follows: (1) acne keloid (dermatitis papillaris capillitii), four cases; (2) keloid of the ear lobe, two cases

(one of these cases is in reality an early hypertrophic scar which began to appear about two months after surgical piercing of the ear for earrings); (3) post-traumatic keloids, ten cases (three cases following incision); (4) post-acne scarring with keloidal formation, eight cases; (5) keloids, cause undetermined (spontaneous?), two cases; (6) cutaneous nodule, one case (nodular subepidermal fibrosis (Michelson), four year old female).

METHOD AND PROCEDURE

The method and procedure are quite simple, and pain involved is minimal to moderate. One cubic centimeter of kutapressin is used per 150 TR units of hyaluronidase. We prepare the solution by injecting the kutapressin directly into the vial containing the hyaluronidase. The resultant mixture is then injected directly into the lesion to be treated in amounts of from 1/10 to 2 cc. (The total amount used is dependent upon the size, age and consistency of the lesion.) The interval between injections varies from four days to two weeks. Again this is dependent upon the aforementioned factors, together with the degree of softening and/or resolution. In a long-standing thickened recalcitrant lesion (acne keloid) we have used a total of 3 cc. of solution at one time.

CONCLUSION

We have endeavored to give a preliminary report on the combined use of two preparations, hyaluronidase and kutapressin,* in a small series of cases. Our reports and studies are by no means complete. We are continuing follow-ups and adding new cases. Our aim is for more carefully planned case studies, including histopathologic examinations, with some types of control.

Finally, we hope this preliminary report will stimulate further interest in this method and lead to its further use in investigations with the hope of bringing to fruition the long-sought solution to this very important problem.

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* Kutapressin supplied by the Kremers-Urban Company, Milwaukee, Wis.

Hand Surgery

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I. INFECTION

PROPER treatment of infection in the hand demands a knowledge of the causative organism, its anatomic localization and some general principles of treatment.

BACTERIOLOGIC FACTORS

The causative organism should be determined in every case by use of aerobic and anaerobic cultures as a basis for correct chemotherapy. Cultures should be repeated periodically in order to detect changes in the bacterial flora consequent to secondary contamination or antibiotic therapy.

Exposure of bacteria to an antibiotic agent may lead to the development of resistance to this antibiotic. Because of the indiscriminate use of various antibiotics by the medical profession, this problem is increasing. For example, Meleney found that 17.8 per cent of coagulase-positive Staphylococcus aureus strains were resistant in 1947–1948, whereas in 1951 this resistance percentage rose to 43 per cent.[1] *In vitro* sensitivity tests are valuable in discovering such resistance, so that a proper choice of antibiotic may be made.

When more than one organism is present in an infection, a mixture of antibiotics may be used as determined by culture and sensitivity studies. However, caution must be taken in administering these combinations since some antibiotics are synergistic to each other and some are antagonistic. In general, synergism exists between two members of the bactericidal antibiotics (penicillin, bacitracin, streptomycin, polymyxin B, neomycin) or between two members of the bacteriostatic group (aureomycin, terramycin,® chloromycetin®). Antagonism may occur when a member of the bactericidal group is administered with one of the bacteriostatic group.[2]

Micrococcus (*Staphylococcus*). This group of organisms produces toxins with necrotizing, hemolytic and leukocidic properties. Coagulase-positive organisms are most apt to be pathogenic. They form relatively avascular abscesses containing pus, necrotic tissue and tremendous numbers of bacteria. Because of the reduced blood supply the amount of antibiotic reaching the site of infection is limited. Organisms captured in these relatively impregnable foci may persist despite high blood concentration of the antibacterial agent.

Many strains inhibit the action of penicillin. There may be two mechanisms for this inhibitory phenomenon. Some organisms can produce penicillinase which destroys penicillin, and others appear to elaborate an essential growth factor whose production is blocked by penicillin.[1]

Besides the local pyogenic manifestation this organism may spread by blood stream invasion, producing either a fulminating or chronic infection. In the fulminating type there is a high temperature and pulse rate with profound toxemia. The usual systemic infection, however, runs a chronic course and is accompanied by metastatic abscesses in the lungs, kidneys, liver, spleen, heart and bones.

Surgical incision still plays a major role in eradicating these infections, but antibiotic therapy is necessary. For local application bacitracin in concentration of 1,000 units per cc appears to be of most value. For systemic therapy one may choose between penicillin, streptomycin, terramycin or erythromycin, depending on sensitivity studies. Bacitracin may be used parenterally if the daily fluid intake is at least 2,500 cc.[1]

Streptococcus. The classification of this organism depends on antigenic substances and

the ability to hemolize blood. Group A, beta-hemolytic strains produce a great majority of streptococcal disease in man, with occasional strains in group C and G being responsible. These three groups produce fibrinolysin, desoxyribonuclease, hyaluranidase and erythrogenic toxin. The non-hemolytic streptococci are usually secondary invaders and usually produce a chronic disease. The anaerobic strains of streptococcus will be discussed later.

The general pattern in hand infections is cellulitis with lymphangitis and lymphadenopathy associated with fever and toxic symptoms. The organisms may enter the blood stream and metastasize to joints, lungs, kidneys, spleen, liver, brain and skin. The endothelial-lined cavities, such as pleura, pericardium, peritoneum and meninges, may also become involved. Penicillin is the antibiotic of choice for the hemolytic streptococci. The non-hemolytic organisms, including enterococci and viridans, may be resistant to this drug and require other antibiotics chosen by sensitivity studies.

Symbiotic Infection. The combination of micro-aerophilic non-hemolytic streptococcus and hemolytic staphylococcus aureus produces a destructive lesion of the skin and subcutaneous tissue. The lesion is usually painful and tender, and the gross appearance is quite characteristic. There is an outer zone of erythema varying from 1 to 2 cm. in width, a dark purple sharply defined middle zone and an inner zone of gangrenous skin. The inner margin is only slightly undermined but gradually melts away as the whole lesion spreads centrifugally. The center of the lesion becomes a granulating ulcer, which in time may become bright red and clean. The disease continues to spread until the patient dies or the infection is brought under control. Chemotherapy is of little avail although Meleney believes that bacitracin is the drug of choice.[3] The usual treatment is wide surgical excision plus the antibiotics which are effective against the causative organisms as demonstrated by sensitivity tests. Activated zinc peroxide may occasionally be beneficial.

A recent tragic experience with this infection is noteworthy: Mr. F. (Boston City Hospital No. 1415513) was a fifty-nine year old white man who sustained a compound fracture of the right thumb while at work. The wound was sutured elsewhere. Five days later on entry to the Boston City Hospital it appeared as demonstrated in Figure 1A. It was believed that this was a severe pyogenic infection, and accordingly penicillin therapy was instituted and an incision made. The incision revealed no pus, and a specimen was sent for culture. The infection continued to spread until, approximately five days after entry, it involved the dorsum and volar aspect of the wrist and lower forearm as demonstrated in Figure 1B. At this time symbiotic infection was diagnosed and bacteriologic studies revealed the presence of enterococci, Staph. aureus, Aerobacter aerogenes, Bacillus proteus. Cultures were sent not only to our own hospital laboratory but also to the laboratory of Dr. Chester Howe to attempt to uncover microaerophilic streptococcus. Aureomycin was instituted intravenously for approximately five days. Localized phlebitis developed at the site of each aureomycin infusion. Infection continued to spread along the forearm on the volar and dorsal surfaces and into the palm and dorsum of the hand as demonstrated in Figure 1C. The treatment so far consisted of wide surgical excision, but it was decided to try parenteral bacitracin. This had to be stopped in approximately four days when the non-protein nitrogen rose to a high level and protein casts were found in the urine. The local agent applied to the excised area was hydrogen peroxide alternating with bacitracin solution. At this stage sensitivity studies, performed by Dr. Chester Howe, revealed the organisms to be sensitive to a combination of streptomycin and sulfamylon.® Following wide excision of the infected area, the wound was dressed with an occlusive dressing under which multiple catheters were applied, and through which the streptomycin-sulfamylon mixture could be injected at numerous intervals during the day so that a high concentration of this agent would be present continuously. Using this approach, the infection appeared to be controlled with a complete denuding of the skin and fascia from the elbow down to the metacarpophalangeal joints of the fingers involving many of the extensor and flexor tendons. The involved area began to granulate and split-thickness skin grafts were applied to the granulating areas.

Approximately four weeks following entry a similar process developed in the right arm at the site of the intravenous aureomycin infusion (Fig. 1D) and over the right deltoid muscle at the site of the penicillin injection. These areas progressed in spite of wide surgical exci-

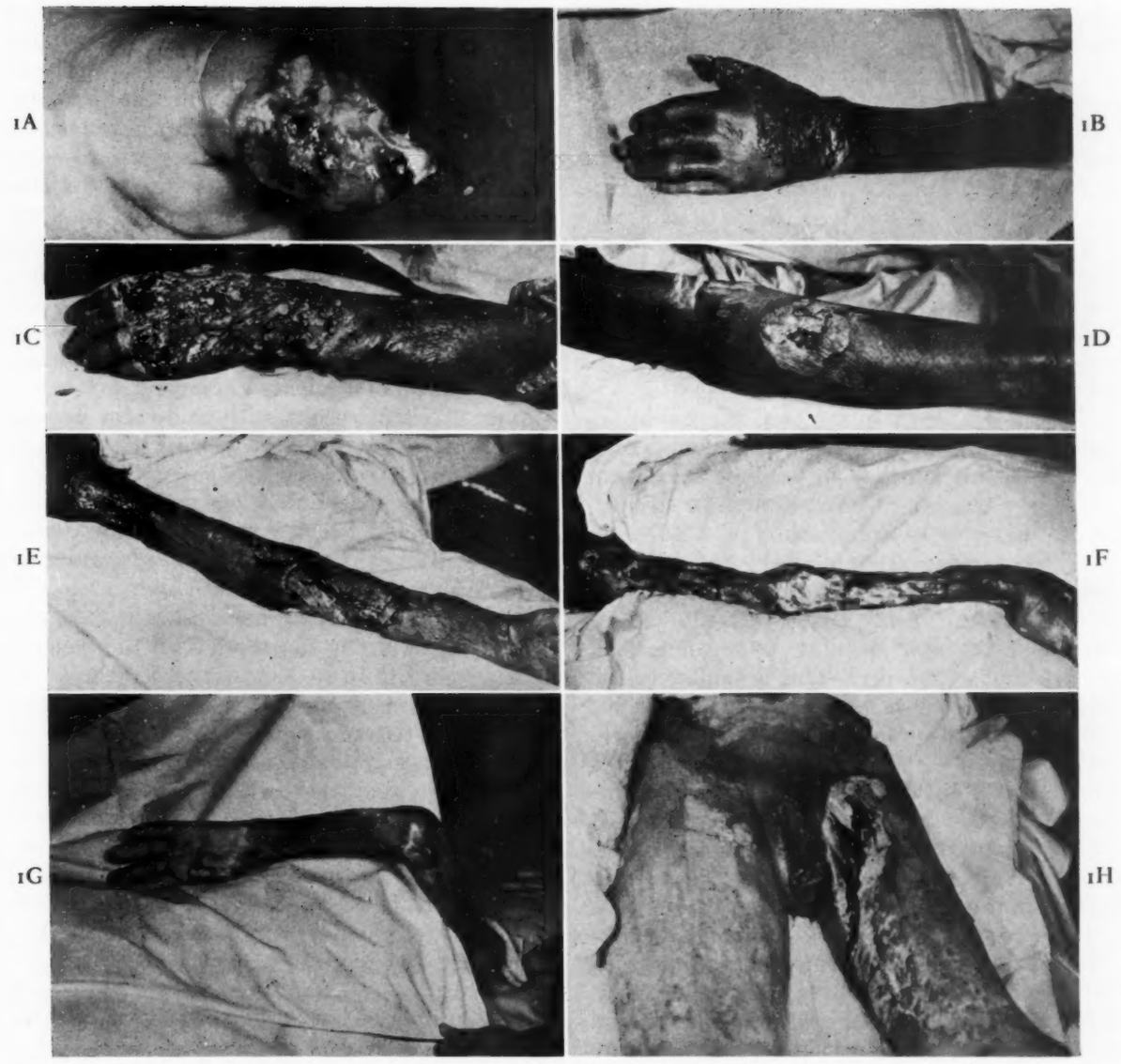


FIG. 1. Symbiotic infection. A, compound fracture, left thumb, five days following injury with development of severe infection. B, spread of infection from thumb to wrist. C, spread of infection three weeks following injury. D, development of symbiotic infection at site of aureomycin injection, right forearm, four weeks following initial injury. E, spread of symbiotic infection in right arm. F, spread of infection from right deltoid to right wrist, with involvement of underlying fascia and tendons. G, skin grafts healed well over site of previous infection in left forearm. H, spread of symbiotic infection to both groins five months following initial infection.

sion and local chemotherapy consisting of streptomycin and sulfamylon (Fig. 1E) until the entire right upper extremity from the deltoid to the wrist was denuded of skin and fascia. (Fig. 1F).

Six weeks following entry, while the right and left arms were progressing as described, massive bilateral deep phlebitis of the lower extremities developed with resultant edema from the knees to the toes. No definitive treatment was instituted for the phlebitis. Anticoagulants were contraindicated because of the extensive ulcerating area, and a surgical attack in the groins could not be done for fear of spread of infection to this area. During the ensuing months of hospital stay the edema gradually subsided and no pulmonary emboli were noted.

Finally, after numerous excisions and ultimate skin grafting, the disease appeared to be

checked about five months following entry and the extremities were covered with skin. Figure 1G shows the left arm now completely covered with split-thickness skin grafts. However, while convalescing from these infections a patch of redness was noted in the left groin which progressed to a similar type of infection in this area that ultimately spread to the right groin. Figure 1H shows the patch of symbiotic infection in the left groin involving skin and fascia with the onset of infection in the right groin. While surgery was being contemplated for this condition, the patient died suddenly in a completely cachectic state.

This case raises many issues. Correct débridement and care of the initial injury should have prevented this infection in the first place. Secondly, the absence of micro-aerophilic streptococcus on repeated examination of cultures by two bacterologic laboratories is of interest. Staph. aureus was the predominant organism which was occasionally mixed with the various bacteria such as Bacillus pyocyaneus, B. proteus and enterococci. This is similar to the experience of Wilson.[4] Thirdly, the reason for the spread of this infection to the opposite arm and both groins is not clear. The involved sites were all areas of previous skin punctures. However, numerous brachial blocks were performed without subsequent local infection. It may be that greater care was taken to sterilize the skin before the brachial blocks than before the routine venipunctures and injections. The phlebitis in the right arm and both legs with its attendant edema may have produced an area of decreased tissue resistance. It is well known that such edematous areas are more liable to infection than normal areas.

Wilson has recently reported a series of somewhat similar cases calling this disease "necrotizing fasciitis." Hemolytic staphylococcus was the most frequent offending agent in this series. The name is derived from the fascia necrosis which is a consistent manifestation of the disease. For surgical excision Wilson lifts skin flaps off the underlying involved fascia, removes the purulent and necrotic fascia for the entire extent of its undermining and then permits the skin flaps to return to their original position. In this way he prevents tremendous skin losses which are the result of the older methods of treatment.[4] It seems to the reviewer that this is a very worth while suggestion.

The *human bite* is another symbiotic infection in which the anaerobic streptococci are the most significant organisms. These lesions also contain pathogenic actinomycetes, fusiform bacilli and spirochetes. When the bite involves the metacarpophalangeal joint, the anatomy predisposes to entrapment of the organisms in the joint.[5] Therapy consists of soap and water cleansing of the wound for twenty minutes; irrigation with sterile saline solution; leaving wound open to heal by secondary intention; applying a sterile dressing; splinting the hand in a position of function; instituting antibiotic therapy. Although most surgeons use penicillin as the antibiotic agent, Levin and Longacre[6] found excellent results with bacitracin in conjunction with the other general methods of treatment.

It is the belief of those on the Boston City Hospital Hand Service that when the metacarpophalangeal joint is involved and the wound seen early, the joint should be opened by means of two lateral incisions, irrigated and left open. A drain may be left in the wound, taking care that it does not enter the joint space but merely keeps the skin edges and underlying tissues separated.

Mycobacterium tuberculosis. This organism may infect tendon sheaths, bones and joints. The tenosynovitis is of special interest and is seen in people who work with cattle (milkers, skinners, butchers, etc.). It is caused by direct extension of an infection in the hand, or it may be secondary to pulmonary tuberculosis. Trauma may be a predisposing factor. The third and fourth decades are the most common age groups involved. The pathology of this disease is the same as other types of tuberculosis. It affects the synovia first, and at a later date it involves the tendon and surrounding structures. The volar tendons are twice as frequently involved as the dorsal ones, and the right upper extremity is afflicted more than the left.

There is a slow onset of a relatively painless swelling over the involved tendon or tendons with eventual limitation of function. At first the swelling is doughy in consistency, but as the disease progresses it may become fluctuant. When it involves the ulnar bursa, it presents an "hour-glass" deformity with a confluent mass in the palm and forearm (compound ganglion). The semi-fluid mass may be pressed back and forth under the volar carpal ligament. If rice bodies are present, crepitus may be felt.

Usually there are no general signs of disease until late in the process.

Treatment consists of complete excision of the involved parts (Fig. 2) plus immobilization in a plaster cast and a course of treatment with streptomycin and para-aminosalicylic acid for approximately six weeks. Bickel et al.[7] in a follow-up study of thirty patients operated upon at the Mayo Clinic found excellent results in eight cases, good in eleven cases, fair in four cases and seven failures which required subsequent surgery on tendons or joints.

Occasionally a tuberculous infection of the skin of the fingers will occur if there is an abrasion or cut which can become infected. Doctors and nurses who work with tuberculous patients are particularly liable to develop this chronic ulcerating lesion.

Girdwood[8] calls attention to Jüngling's disease or multiple cystic tuberculosis of bone. There is a gradual onset of swelling in a small bone of the hand or foot which may be moderately painful. X-ray shows a cystic trabeculated expansion of the bone. The disease takes a slow course tending to spontaneous improvement and even recovery.

Treponema pallidum. This organism rarely involves the hand, but chancres may occur at the site of a hangnail and be mistaken for paronychia. Tertiary lues may affect the tendon sheaths, bones and joints of the fingers and wrist. Discussion of the treatment of this condition is beyond the scope of this review.

Pasteurella tularensis. This organism produces infectious disease in rodents and in other wild and domesticated animals such as game birds and domestic chickens. Man becomes infected with this organism when he is bitten by an insect vector (tick or deer fly) or when he comes in contact with diseased animals. If the source of entry is the skin of the hand, a papule-like lesion develops which later ulcerates. A red granuloma occurs with a surrounding zone of cellulitis. Later, the center of this becomes necrotic leaving a punched-out ulcer. Bacteria spread along the lymphatics with resultant lymphangitis and lymphadenitis. In half the cases the glands suppurate. Septicemia may develop with foci of infection in the spleen, liver, lungs, lymph nodes, bone marrow or other tissues. Identification is made by bacterial smear, direct culture, inoculation in guinea pigs and the Foshay intradermal test. Positive blood cultures may be obtained during the first week, and agglutinins are demonstrable after ten days. Streptomycin is effective in eradicating this disease. There is some evidence that aureomycin and chloramphenicol are effective.

FIG. 2. Tuberculous tenosynovitis of left wrist showing infected tissue being dissected from underlying tendons.

Bacillus anthracis. This aerobic spore-forming organism causes disease in sheep, cattle, horses and swine. Human infection develops from handling such animals on the farm or from manufacturing and processing their products in the factory and canneries. A red macule develops that enlarges and produces a vesicle which ruptures, leaving a characteristic black gangrenous base. There may be surrounding vesicles. The lymph glands will become enlarged and tender with associated malaise and fever. The disease may produce septicemia with subsequent pneumonia, endocarditis and meningitis. Diagnosis is obtained by inoculation of mice with subsequent culture of the organism and observation of a Giemsa-stained smear of the spleen. Occasionally a direct microscopic examination of a smear from the lesion may give a tentative diagnosis. Agglutination tests with B. anthracis are not satisfactory. Penicillin therapy appears to be very effective.

Malleomyces mallei. This organism produces a rare disease in which the infected mucus from horses, mules or donkeys enters an abrasion of the hand causing an initial lesion that is called "farcy." A granulomatous lesion develops which ulcerates and becomes encircled by small vesicles and pustules with associated lymphangitis and lymphadenitis. The ulceration may spread into the muscles, or infection may spread to the lungs, perito-

neum, skin, joints and bones. Diagnosis is by smear, culture and guinea pig inoculation. There is also a subcutaneous mallein test, an agglutination test and a complement-fixation test.

Neisseria gonorrhoeae. This produces tenosynovitis of various tendon sheaths or arthritis of the wrist and finger joints. The acute tenosynovitis may be difficult to diagnose from an ordinary pyogenic infection. The diagnosis is by smear and culture of the infected tissue. Penicillin is the antibiotic of choice.

Erysipelothrix rhusiopathiae. This organism is found in a wide range of animals and produces the swine erysipelas. It survives in decomposing nitrogenous material and is often found in decomposing fish, shellfish or meat. It produces infection in man by penetrating the skin of the hand through a wound or abrasion. This is most commonly found in abattoir workers and fish handlers.[9] There is severe erythema and edema of the part with pain persisting in the affected area. The infection is self-limited and seldom goes beyond the wrist. Relapses are common and occasionally a generalized septicemia with infection of the endocardium and joints may occur. Diagnosis is by biopsy and culture. The treatment is conservative since it is usually self-limited. Penicillin appears to be the antibiotic of choice.[10]

Actinomyces bovis. Frequently cattle, swine or horse workers may be invaded by this organism through an abrasion or wound of the hand. There is a granuloma similar to that of tuberculosis which will remain for months as an indolent nodule with occasional sinus formation. It may cover the entire dorsum of the hand. Diagnosis is made by microscopic examination and culture of the draining material. Penicillin and aureomycin may be of benefit in treatment. Isoniazid (nydrazid®) has recently been reported as beneficial.[11]

Blastomyces dermatitidis. Infection of an abrasion or wound of the hand by this organism produces a papulopustule which spreads peripherally. The center may heal, leaving a scar surrounded by a spreading border. A systemic type of disease beginning in the lungs with a hematogenous spread to the skin and bones may occur. Diagnosis is made by smear and culture of the draining material. Iodides and x-ray have been advised for treatment.

Sporotrichum schenkii. This widely distributed fungus may infect the extremities through abrasions or thorn pricks. A pustule or abscess develops with invasion of regional lymphatics producing a thickening of the lymph vessels with multiple granulomas along the course of these vessels which may rupture spontaneously. Diagnosis is made by examination and culture of the draining material. Treatment with iodides may be of value.

Coccidioides immitus. This organism rarely affects the hands. When it does, it causes skin nodules or ulcers. Diagnosis is made by examination of smear and culture.

Monosporium apiospermum. This organism which causes madura foot occasionally affects the hands, giving rise to a granulomatous lesion similar to actinomycosis. Diagnosis is made by smear, culture or biopsy. Although antibiotics are of little value, they may prevent secondary infection.

ANATOMIC LOCALIZATION

Suppuration in the hand must be treated by incision and drainage. Knowledge of the various anatomic spaces is essential for this purpose. Incisions must be placed to drain pus adequately, but they should avoid tactile surfaces of the fingers and palm whenever possible. They should also avoid crossing flexion lines of fingers or palm and should not damage underlying structures.

Pulp Spaces. A *felon* is an abscess of the pulp space of the terminal digit manifested by a painful, swollen, indurated and tender fingertip. If not treated early, osteitis and skin necrosis may occur. The skin necrosis not only results in a deformed distal phalanx but also greatly increases the healing time.

Extension of the infection shuts off the blood supply to the diaphysis of the terminal phalanx causing aseptic necrosis of bone which may become secondarily infected. Bolton et al.[12] challenge this concept of occlusion of the digital vessel and believe that bone involvement is due to direct extension of infection to the periosteum. Probably both mechanisms play a role in producing osteitis. X-ray evidence of osteitis is of prognostic value. Robins[13] found the healing time of simple felons to be ten days; whereas if osteitis was present, the healing time was thirty days.

Treatment consists of wide excision around the end of the finger taking care that the incision passes almost underneath the nail.

(Fig. 3.) The usual textbook of surgery demonstrates this incision carried across the tip of the finger rather than dorsal to it. This may not only leave a painful scar in an important tactile area but also may result in a deformed fingertip due to sloughing of the skin dorsal to

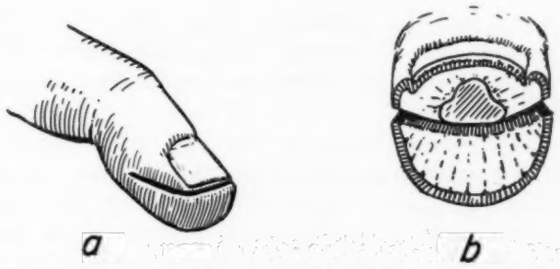


FIG. 3. Incision of felon. (*a*) Anterior incision should be placed close to nail to avoid scar on tactile surface. (*b*) Incision should be carried through all fibrous septa of distal pulp space.

FIG. 4. Poorly placed incision for felon producing deformed fingertip.

the incision. (Fig. 4.) The fibrous septa, from skin to periosteum, must be cut through completely and all necrotic tissue excised. If loose bone is found in the pulp, it should be removed. If the wound does not heal in several weeks, the possibility of sequestration should be entertained and additional x-rays taken. If sequestration is found, it can be remedied by surgery.

Scott and Jones[14] have reviewed their experiences with excision of necrotic tissue and primary suture as a method of treatment of surgical infections of the hand. Primary healing occurred in 56 per cent of 226 felons, with an average healing time of eleven days. The average healing time of the other cases was thirty-four days. It is difficult to see the advantages of this method of treatment, since in Robins' series of 197 felons in which a conventional "hockey-stick" incision was made, the healing time was ten days for simple cases, fifteen days with skin necrosis and thirty-two days with osteitis.[13] In other words, when primary healing takes place by this immediate suture method, the average healing time is about the same as for the standard drainage methods. If it does not occur, healing time is prolonged.

Two other infections of the distal phalanx may occur which are not as serious as true felons. One is the *apical abscess* which lies in the distal pulp space under the nail bed and anterior to the phalanx.[15] Osteitis seldom occurs with this, and hence the average healing time is short. Treatment consists of removal of skin and distal portion of nail to provide free drainage. The other infection is a *subepithelial abscess* in which the underlying pus can be seen through the thin wall and requires merely an unroofing without anesthesia. It may mask an underlying deep abscess, in which case a sinus tract can be demonstrated.

Similar infections can occur in the pulp spaces of the proximal and middle phalanges and must be surgically drained. One incision is a right angle one which is carried through the digital crease and extended along the lateral aspect of the finger. When infection is present in the pulp space of the proximal phalanx of the finger, Iselin[16] suggests making an incision which is carried along the lateral aspect of the finger to the web space which is then completely opened by an anterior-posterior incision. This may result in an adduction deformity.

Paronychia. They begin in one portion of the paronychium or eponychium, and treatment consists of a simple incision of the abscess. When left untreated, however, they may run completely around and under the nail as a "horse-shoe" shaped abscess. Drainage must then be accomplished by lifting a flap of eponychium and removing the proximal half of the nail. In developing the eponychial flap, two incisions are made at the lateral angles of the nail bed distal to the lunula so as to avoid damaging the nail matrix. They are extended lateroproximally. (Fig. 5.)

Chronic paronychia should make one suspicious of fungus infections which are quite

common in people whose hands are frequently immersed in water.

Web Spaces. In the web spaces between the fingers, compartments are formed which are bounded on either side by the pretendinous palmar fascia bands, proximally by the superficial transverse palmar ligament, distally by the natatory or intradigital ligament, and overlain volarly by a small fat pad. The web space contains the digital nerve and arteries and the lumbrical muscle. The tissue in this space is the common site of infection from skin abrasions, calluses or splinters. The infection points to the palmar aspect of the web space but may spread dorsally by lymphatic invasion or direct spread. Infection may also extend to the proximal phalanx or along the lumbrical canal to one of the palmar spaces. If the abscess is confined to the palmar surface, a small curving incision over the interspace will be sufficient. (Fig. 6A.) However, with dorsal extension, a longitudinal incision on the dorsum of the web space may be necessary. (Fig. 7A.) The web should not be incised completely since this might lead to an adduction contracture of the fingers.

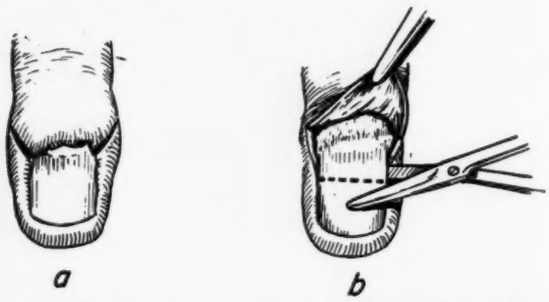


FIG. 5. Incision of paronychia. (*a*) Lateral incisions should begin distal to lunula. (*b*) Proximal half of nail removed after eponychial flap is raised.

Occasionally, persons dealing with hair or hides develop lesions produced by short hairs working into the skin and stimulating a foreign body reaction. In the dairy industry these are known as "milker's granuloma." Downing[17] has reported such a lesion resembling a pilonidal sinus appearing in the web space of barbers.

Superficial Palmar Fat Pads. In the palm of the hand there are fat pads over the thenar and hypothenar eminences which may be the site of an abscess. These may be confused with some of the more important palmar spaces. Treatment is incision over the area of fluctuation.

Thenar and Hypothenar Muscle Spaces. The fascia covering the hypothenar and thenar muscle groups forms potential spaces which

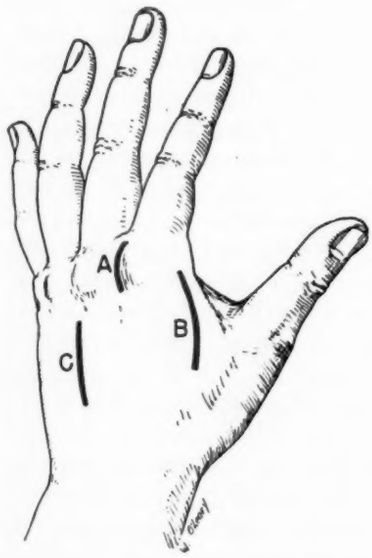


FIG. 6. Incisions for draining infections of the hand. A, web space; B, midpalmar; C and C′, quadrilateral space of the forearm; D and E, digital tenosynovitis; F, radial bursa; G, ulnar bursa.

FIG. 7. Incisions for hand infection. A, web space; B, thenar space; C, dorsal space.

may be the site of abscesses. These are drained by suitable incisions over the area of maximum tenderness and fluctuation. Care must be taken to drain the thenar muscle space abscess at some point distant from the motor branch of the median nerve. This point has been well described by Kaplan[18] using the cardinal line as a landmark. This line is drawn from the apex of the interdigital fold, between the thumb and index finger, parallel with the middle crease of the palm of the hand, passing near the distal pole of the pisiform bone. The intersection of this line with the thenar crease corresponds to the underlying motor branch of the median nerve.

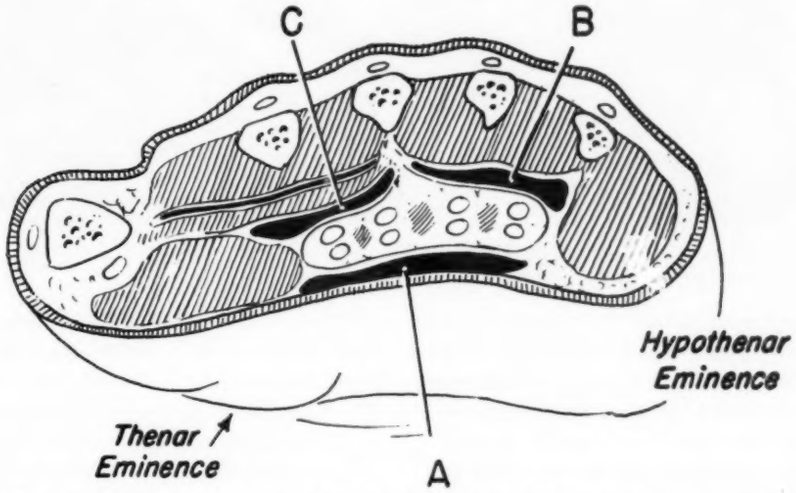


FIG. 8. Schematic cross section of palm of hand. A, pretendinous space of Iselin; B, mid-palmar space; C, thenar space.

Palmar Space. The palmar space is bound laterally by the thenar and hypothenar eminences, dorsally by the interosseus muscles and fascia, volarly by the palmar fascia, proximally by the apex of the palmar fascia where it joins the transverse carpal ligament, and distally where the palmar fascia ends as pretendinous bands to the fingers or at the superficial transverse ligament just proximal to the web spaces. In this compartment lie the flexor tendons to the four fingers surrounded by the ulnar bursa as well as the digital nerves, vessels and lumbrical muscles. This ulnar bursa with enclosed tendons divides the palmar space into a pretendinous space and a retrotendinous space.

The pretendinous palmar space has been well described by Iselin.[16] Figure 8A demonstrates this space on transverse section of the palm. Clinically there is a central swelling of the palm with well marked lateral limitations. There is the usual red edema of the dorsum of the hand. This space may be drained by a transverse incision in the palm paralleling the proximal palmar crease. (Fig. 6B.)

There has been great controversy concerning the retrotendinous palmar space. Most authorities agree that it is divided into two by a septum of fascia arising from the undersurface of the flexor tendons and attached to the entire length of the middle metacarpal bone.[19] Kaplan[18] believes that this septum is formed by the adherence of the ulnar bursa to the interosseus and adductor fascia in the region of the crest of the third metacarpal where the transverse fibers of the adductor pollicis muscle take origin. Regardless of the origin of this septum, it divides the retrotendinous compartment into the middle palmar and the thenar palmar spaces of Kanavel.[20] Figure 8B and C demonstrates these compartments in a transverse section of the palm. A fat pad is almost universally present in the mid-palmar space of the hand which may be a reason for the vulnerability of this space to infection.[18]

The *mid-palmar space* abscess is manifested by swelling and tenderness in the central palm. The third, fourth and fifth fingers may be held in partial flexion and there is the usual dorsal edema associated with severe hand infections. It may be drained satisfactorily by a transverse incision through or parallel to the distal transverse palmar crease. (Fig. 6B.)

FIG. 9. Tenosynovitis of right index finger demonstrating characteristic position of finger.

FIG. 10. Incision for tenosynovitis of right index finger.

The *thenar space* should not be confused with the thenar muscle space. Since the floor of the thenar space is the adductor pollicis muscle and fascia, it is believed that a better term for this space would be the "adductor space." Infection in this area may extend around the free border of the adductor muscle to the dorsum of the web space between the thumb and index finger resembling a web space infection. A thenar space abscess presents tenderness and swelling over the thenar portion of the palm with much edema in the web space between the thumb and index finger. There is usually partial flexion and limitation of motion of the index finger, and the thumb is held in abduction. This space is drained by an incision in the web space of the thumb on the dorsal aspect parallel with the anterior surface of the second metacarpal bone. (Fig. 7B.) The first dorsal interosseus muscle is retracted ulnarly and dissection is continued along the course of the adductor pollicis muscle until pus is reached.

Dorsal Spaces. There are two dorsal spaces in the hand: the *subcutaneous space* between the skin and the extensor tendons and their intertendinous fascia, and the dorsal *subaponeurotic space* between the extensor tendons and the metacarpal bones and interosseus muscles. Either space may be drained by a longitudinal incision, taking care to avoid the underlying tendons. (Fig. 7C.)

Carbuncles occur most frequently on the dorsum of the first phalanx of the finger, although they may appear anywhere on the hand. The principles of treatment of carbuncles in other parts of the body are applicable to the hand. With modern chemotherapy surgery is usually limited to minor incisions of pyogenic cavities. X-ray may help in localizing the infection. Beware of diabetes!

Quadrilateral Space of the Forearm (Parona). This space is bound dorsally by the pronator quadratus muscle and the interosseus membrane of the forearm, volarly by the flexor digitorum profundus tendons; radially by the flexor carpi radialis, and ulnarly by the flexor carpi ulnaris. Distally it communicates with the carpal canal where it has a relation with the radial and ulnar bursae. Infection may spread to the quadrilateral space from the palmar spaces or from the radial and ulnar bursae. Clinically, there is swelling and tenderness of the entire volar aspect of the forearm. The abscess may be drained by a longitudinal incision parallel with the medial borders of the radius or ulna, taking care to avoid the underlying structures. (Fig. 6C and C′.)

Tendon Sheath. A knowledge of tendon sheath anatomy is a basic requirement for correct surgical treatment of tenosynovitis. The tendon sheaths of the index, middle and ring fingers extend from the insertion of the flexor profundus tendon in the distal phalanx to the mid-palmar creases. The tendon sheath of the thumb begins at the insertion of the flexor pollicis longus tendon in the distal phalanx and extends into the wrist through the carpal canal. The extension of this tendon sheath into the palm is called the "radial bursa." The tendon sheath of the fifth finger extends from the insertion of the profundus tendon in the distal phalanx to the wrist under the carpal tunnel. The portion of this tendon sheath in

the palm is called the "ulnar bursa" and it enlarges to invest all the flexor tendons of the fingers. The ulnar bursa is arranged in three communicating compartments: superficial, anterior to the tendon of the flexor sublimis; middle, between the tendons of the sublimis and profundus; posterior, dorsal to the tendon of the flexor profundus. The lumbrical muscles are outside of the ulnar bursa. This arrangement is present in approximately 70 per cent of investigated cases.[21]

The cardinal points of digital tenosynovitis which have been emphasized by Kanavel[20] are: tenderness over the involved tendon sheaths, pain on hyperextending the finger, flexion deformity of the involved finger and swelling of the involved part. Figure 9 demonstrates a typical case of digital tenosynovitis. In order to distinguish tenosynovitis from subcutaneous cellulitis, Moses[22] uses the following test: "The nail of the involved finger is fixed by the examiner so that actual flexion will not be allowed. The patient is then to attempt flexion and if tenosynovitis is present, this will cause pain. Since, however, the finger does not move, a cellulitis will not produce pain."

Tenosynovitis should be incised as early as possible in the course of the disease. In the finger the most useful incision is a mid-lateral one, extending the entire length of the finger. The ends of the digital creases serve as landmarks for the lateral plane of this incision. (Figs. 6D and 10.) It is also necessary to make a curved incision over the palmar cul-de-sac of the tendon sheath. (Fig. 6E.)

When the *radial bursa* is involved, there will be the usual signs of tenosynovitis in the thumb associated with tenderness along the course of the bursa. In addition to the usual digital incision on the thumb, the bursa may be drained by a curved incision over the ulnar aspect of the thenar eminence taking care to avoid the underlying motor branch of the median nerve. (Fig. 6F.) If the volar carpal ligament must be incised to drain this tendon adequately, it must be opened on its lateral side rather than in the middle to avoid prolapsing of tendons. The cul-de-sac of the radial bursa in the wrist is found and drained through a longitudinal incision immediately ulnar to the distal 3 inches of the flexor carpi radialis. (Fig. 6C'.)

Infection in the *ulnar bursa* presents a swelling on the palmar and dorsal surfaces. There is tenderness along the course of the tendon sheath of the fifth finger and along the ulnar bursa. There may be tenderness just proximal to the transverse carpal ligament in the wrist. All the fingers are held semi-flexed and pain is produced on attempting passive extension. There is limitation of motion of the wrist. The incisions to be used in opening this infection are a mid-lateral incision along the fifth finger, a curving palmar incision which just skirts the hypothenar eminence (Fig. 6G) and an incision just radial to the distal 3 inches of the ulna. (Fig. 6C.) The fascia of the forearm is incised in line with the forearm incision, and the flexor carpi ulnaris is retracted radially. The pronator quadratus muscle is exposed, and lying on its distal portion is the bulging cul-de-sac of the ulnar bursa which is incised. If the bursa has ruptured into the quadrilateral space of the forearm, a counterincision is made on the opposite side of the forearm.

Joint Spaces. These spaces may become infected by trauma, spread from neighboring infections or metastasis. The most common organisms are streptococcus, staphylococcus and rarely gonococcus.

The *interpbalangeal* and *metacarpopbalangeal* joints have a fairly similar pattern of anatomy. Each possesses a capsule which is reinforced laterally by the lateral collateral ligaments running in a volar direction from the proximal bone to the distal bone. The volar aspect of the joint is reinforced by a strong ligament known as the "volar accessory ligament" or the "palmar plate." The fibrous tendon tunnels adhere to the palmar plates. The dorsum of the joints are reinforced by various portions of the extensor mechanism, the distal interphalangeal joint being reinforced by the terminal insertion of the extensor apparatus of the finger and the proximal interphalangeal joint being reinforced by the central extensor tendon and lateral band. Kaplan[18] found loose connections present in the metacarpophalangeal joint between the extensor apparatus and the dorsal aspect of the capsule in 38 per cent of a series of cases.

Infection in the digital and metacarpophalangeal joints may present swelling, redness, tenderness, excessive mobility and crepitus. X-ray will reveal the disease by the second or third week when decalcification and erosion of the joint line may be seen. When the infection spreads beyond the joint, it tends to spread dorsolaterally since the capsule is most thin

at this point. Rarely, the infection may spread anteriorly into the flexor sheaths, particularly at the proximal interphalangeal joint. Treatment is by chemotherapy, surgical incision and immobilization in the position of function. Usually two lateral incisions over each joint are sufficient for adequate drainage. Motion should be started as soon as the infection has subsided.

The *wrist* includes the following joints: the distal radio-ulnar, radiocarpal, proximal intercarpal, distal intercarpal, mid-carpal and carpometacarpal. The more important joints are the radiocarpal and the mid-carpal through which most of the motion in the hand occurs. Kaplan[18] has summarized the motions of the wrist approximately as follows: ". . . volar flexion of the hand takes place mostly in the radio-carpal joint and secondarily in the mid-carpal joint. Dorsiflexion, on the contrary, occurs mostly in the mid-carpal joint and additionally in the radio-carpal joint. Radial deviation occurs mostly in the mid-carpal joint, and ulnar deviation occurs mostly in the radio-carpal articulation.

Infection of the wrist joint is demonstrated by swelling, pain and tenderness in the region of the wrist. A persistent swelling on the dorsum of the wrist is fairly pathognomonic. Crepitus in this joint is a late manifestation. X-ray taken during the second or third week of the disease will show disintegration of the carpal bones. The principles of treatment are the same with the wrist joint as in the other joints. The incision is usually performed over the ulnar side of the wrist as recommended by Bunnell.[23] Through a lateral incision over the joint, the tendons of the flexor and carpi ulnaris are severed at their insertions. The wrist joint can be widely opened by cutting the joint capsule, and necrotic carpal bones removed if necessary. The head of the ulna should be removed to permit pronation and supination. Immobilization should include the upper arm to prevent supination and pronation. The position of the wrist should be mid-way between supination and pronation with about 15 degrees dorsiflexion.

Lymphatic System. The superficial collecting lymphatics of the fingers continue laterally to the base of the fingers where they pass through the interdigital cleft to reach the dorsum of the hand.[24] The lymphatic trunks of the palm pass in four directions: inferiorly, into the web space of the finger to ascend on the dorsum of the hand; laterally and medially, around the border of the hand to unite with the lymphatics on the dorsum of the hand; superiorly, toward the anterior surface of the forearm.

The collecting trunks of the dorsum of the hand continue upward along the posterior surface of the forearm, and they incline toward their adjacent borders of the forearm just above the wrist. The lateral collecting trunks, from the thumb and index finger, and the medial collecting trunks turn around their adjacent borders of the forearm and continue up the arm alongside the anterior collecting trunks from the palm of the hand. The medial, anterior and lateral collecting trunks of the forearm continue along the anterior surface of the arm to terminate in the axillary nodes chiefly in the axillary vein group and in the central group. A few of the lateral collecting trunks in the upper part of the arm course along the cephalic vein and deltopectoral sulcus to terminate in the subclavicular or supraclavicular nodes.

The deep lymphatics begin in the fingers, continue along the digital vessels and course along the posterior interosseus and palmar arches up the forearm accompanying the large vessels, such as the ulnar, radial, anterior and posterior interossei. Each of these blood vessels is accompanied by two lymph trunks which anastomose frequently.

In the arm these vessels course along the brachial and radial arteries and terminate in the axillary lymph nodes, chiefly in the axillary vein group and the central group.

Inflammation of the lymphatics is usually caused by streptococcus, but many other organisms spread in this fashion. Clinically, lymphangitis demonstrates a high temperature, tachycardia and malaise associated with red streaks along the course of the lymphatic vessels. Abscesses may develop along the course of the lymphatics in the intercalated nodes. Treatment of lymphangitis consists of immobilization and chemotherapy.

GENERAL PRINCIPLES OF TREATMENT

Treatment of hand infections must be carried out as early as possible to prevent spread of infection and irreversible sequelae.

The function of drains is to keep incisions open. They should never plug the wound or prevent a free flow of exudate. They should

never extend into tendon sheaths or joint spaces. Vaseline strips or small rubber strips are the usual drains used. In the fingers it is better to use small rubber strips by choice because of lack of space.

All operations are carried out in a bloodless field with good lighting and assistance under general or regional anesthesia.

Immobilization of the part is essential for recovery. This may be obtained by a massive dressing in the early stages of acute infection; this is later replaced by a plaster of paris splint. When the hand is immobilized for more than a day, it should be placed in the position of function, i.e., the wrist in 20 degrees dorsiflexion, the finger joints in slight flexion, the thumb in abduction and opposition with its interphalangeal joints slightly flexed.

Systemic diseases should be searched for in all cases, with particular emphasis on diabetes, lues or the various bleeding diatheses. The patient's nutrition should be maintained in as normal a state as possible, with particular attention to correction of anemia, vitamin C deficiency and low serum protein. Although many people are being treated with cortisone today, one should remember that wounds and infections seem to heal more slowly when this is administered.

The aftercare of each wound is designed to prevent saprophytic infection. Débridement of dead tissue should be performed whenever it occurs, and clean granulation tissue should be skin-grafted as soon as possible to prevent fibrosis. The subsequent changes of dressings should be carried out under aseptic conditions using sterile instruments and surgical face masks.

Physiotherapy in the form of active and passive motion should be instituted as soon as possible to prevent ankylosis of the finger joints. If a hand has been immobilized for any length of time, physiotherapy should be directed to the elbow and shoulders. In the older age group, these joints may become ankylosed within a relatively short period of time and the subsequent disability may outweigh the hand disability.

II. TRAUMA

The treatment of specific injuries to the hand will be discussed in the appropriate sections of this part but a few general principles concerning the care of the compound injured hand will be prefatorily presented.

All severe lacerations of the hand require care in the operating room. An adequate history and physical examination should be performed, so that any systemic disease may be discovered. The history is of great importance in ascertaining factors essential to therapy, such as the causative agent, place of injury, circumstances under which it took place, type of first-aid treatment and the time from injury to hospital admission. A detailed examination of the hand and arm is begun before operation, with a sterile dressing applied to the wound. This examination should include not only motion but also careful testing for sensation. The type of wound should also be noted since it is well known that incised wounds heal better than crushed or avulsed wounds.

All patients are treated under general anesthesia or regional anesthesia (brachial block). All severe injuries are repaired in a bloodless field obtained by application of a tourniquet to the upper arm. During the operation the tourniquet should be released every hour to prevent ischemic changes in the nerves or other soft tissues of the arm.

The surgeon puts on a cap and mask, scrubs his hands and dons a sterile gown and gloves. The injured hand is shaved from elbow to fingertips and washed for twenty minutes with sapo mollis. The surgeon changes gown and gloves (if there is another surgeon on the case, he takes over) and the wound is irrigated with 5 or 10 L. of sterile saline solution, depending upon the degree of injury. Following this procedure the surgeon changes his gown and gloves, and the wound is draped for operation. Débridement of the wound is done, with all dead and devitalized tissue being removed. The detailed treatment of the various structures will be listed in subsequent sections of this part.

Whether primary or delayed suture is performed will depend on the type of injury, the cleanliness of the wound and the time interval between injury and treatment.

A brief résumé of the treatment of such wounds by the Boston City Hospital Hand Service is as follows: Primary suture of clean incised wounds is done within twenty-four

hours when there is no inflammation. Primary suture of dirty incised wounds, clean avulsed and crushed wounds, and dirty avulsed and crushed wounds—if débridement is complete—can be performed within twelve hours if the wound shows no inflammation. When the

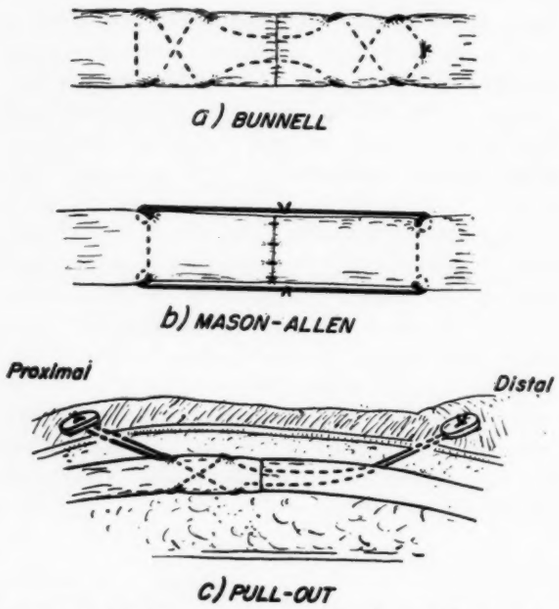


FIG. 11. Various suture technics for tendon repair. (*a*) Bunnell silk suture technic; (*b*) Mason-Allen silk suture technic; (*c*) stainless steel wire pull-out technic (Bunnell).

débridement is incomplete in avulsed and crushed wounds, delayed closure of the wound should be performed. Using this system of treating wound injuries over a three-year period, postoperative sepsis developed in only five of 618 patients.[25]

When the wound has been closed, the hand is placed in a plaster of paris cast in a position of function whenever this is possible. Tetanus antitoxin or tetanus toxoid is given. Penicillin has become routine for the first few postoperative days. The patient is hospitalized for all serious injuries until one is sure that infection is not present in the wound.

TENDONS

Tendon Healing. When lacerated tendon ends are approximated, there is an outpouring of fibrin, during the first two days following injury, which joins the tendon ends. By the fifth day there is an outgrowth of fibroblasts from the connective tissue elements of the tendon into this mass of fibrin. At the end of the second week tendon fibers and cells begin to bridge the gap. During the third week there is formation of strong tendon collagen fibers. The edema and vascularity at the tendon juncture gradually diminish, so that by the end of the fourth week there is good strength in the junction.

There have been numerous studies during the past few years on the prevention of adhesions about tendons during recovery. Weckess et al.[26] found that film material such as human fibrin film, bovine fibrin film and cellophane were effective in preventing adherence of the tendons.

Gonzalez[27] found that the use of polyethylene tubes could prevent adhesions around a tendon anastomosis. However, the tendon required approximately forty days for good healing, since the film wrapped around the tendon interfered with the blood supply at the tendon junction. This would limit the procedure for use in humans, since forty days' immobilization would stiffen the finger joints so that function would be greatly limited.

Grant[28] studied the affect of cortisone on tendon healing and could find no difference in the control and cortisone-treated animals with regard to adhesion formation. A similar study was made by Gonzalez[29] who could find no evidence that cortisone improved or interfered with the healing of wounds.

Methods of Repair. The exact type of repair does not appear to affect the end result too much providing care is taken with the method used. Numerous methods are available using silk or pull-out wire sutures, according to the technics of Bunnell[23] or Mason and Allen.[30] Figure 11 demonstrates several of these suture methods. In general, on the Hand Service at the Boston City Hospital the Bunnell silk suture technic is used for tendon-to-tendon repair, and the wire pull-out technic for tendon-to-bone repair.

Other modifications of tendon repair have been devised. Jennings has recently used a barbed braided tantalum® suture which is threaded through the proximal and distal tendons.[31] A needle is attached to both ends of the suture. The distal needle is threaded through the tendon junction and brought out through the skin. Adequate tension is placed on this suture which is maintained by means of a button. The proximal portion of the suture is threaded through the skin loosely and applied

over a button. A V-shaped steel prong applied in such a way that the prongs point toward the distal needle holds the tendon in place. At the completion of healing the suture is removed by cutting the distal wire and withdrawing the suture by means of the proximal wire similar to the pull-out technic of Bunnell. Those on the Hand Service at the Boston City Hospital have used this suture in a few cases but do not believe that it offers any particular advantage over more conventional methods of suture.

The return of function is usually excellent in the extensor and flexor tendons when they are incised in the forearm, wrist or proximal palm. With regard to the extensor tendons, many authors state that a simple figure-of-eight suture is sufficient providing the wrist and digits are placed in dorsiflexion for three weeks. They state that the extensor tendons so interdigitate that splinting alone often suffices.[23] In two cases of which the author has knowledge the extensor tendons did not so interdigitate and the proximal ends retracted, pulling the suture line apart. It is believed that it is better to suture the extensor tendons as all other tendons are sutured.

Flexor tendons repaired within the digital sheath or tunnel have a poor prognosis. This "danger zone" is the area from the palmar crease to the middle phalanx of each finger. In this narrow fibrous tunnel there are two closely adherent tendons sliding over each other. Most attempts at repairing both these tendons result in adhesion of the tendons to the tunnel with marked limitation of motion of the finger. When the flexor digitorum sublimis alone is divided, it is probably wiser to excise the distal portion to a few millimeters from its attachment to the middle phalanx and the proximal portion, so that it will retract to the palm. Early motion of the fingers should be instituted. The suture of one slip of the sublimis tendon should likewise not be attempted. If both sublimis and profundus tendons are involved, the sublimis should be cut away as previously described and the profundus sutured. The annular ligament in the region of the tendon juncture should be excised completely as suggested by Mason.[32]

There is an increasing tendency on the part of surgeons to treat these injuries in the digital tunnels as primary skin wounds and do a delayed tendon grafting at a subsequent date. Some surgeons are even attempting a primary tendon graft as initial treatment.[33] This latter procedure should be viewed with caution, since if it fails and infection develops in the primary tendon graft it may be impossible to repair the finger.

If the profundus tendon is severed at the level of the middle phalanx, it may be advantageous to excise the distal portion and advance the proximal end into the distal phalanx of the finger. In this way there will be no suture line in the digital tunnel. If there is a contracture of the tendon, it may be stretched by dynamic splinting.

Immobilization. Repaired tendons are immobilized in a plaster of paris cast which is applied so that there is no tension on the tendon junction. The flexor tendons are immobilized for three weeks in a position of slight wrist flexion with flexion of the involved fingers. The weak extensor tendons are immobilized for five weeks, usually with the wrist dorsiflexed about 15 degrees, the metacarpophalangeal joints fully extended with slight flexion permissible in the proximal interphalangeal joints. If there has been loss of tendon, it may be necessary to flex or dorsiflex the hand much more than this. As a rule, only injured digits should be immobilized. In children and irresponsible patients the plaster cast should be made sufficiently strong in order to prevent breakage.

Aftercare. When the cast and sutures are removed, physiotherapy should be started. Heat is of benefit, but the best physiotherapy is active and passive motion of the involved joints until there is a maximum return of function. The wrist, elbow and shoulder of the involved extremity should be similarly treated. In particular, attention should be directed to the shoulder and motion should be begun as soon as possible even while the plaster of paris cast is on the hand. Too often the hand injury recovers but the limitation of motion of the shoulder joint may be severely incapacitating.

Ruptured Tendons. A sudden forced flexion of the distal interphalangeal joint will produce an avulsion of the extensor tendon from the distal phalanx, producing a marked flexion deformity of the distal phalanx. (Fig. 12.) This is known as baseball or mallet finger. Conservative therapy is employed, with the distal interphalangeal joint immobilized in a position of marked dorsiflexion for six to eight weeks. Although the distal joint is kept in dorsiflexion, the proximal interphalangeal joint should be

kept in a position of 15 degrees flexion. This position is maintained by a plaster of paris cast. A Kirschner wire inserted through the distal, middle and proximal phalanges may be used to maintain the same position.[34] If seen late after an injury, operation will probably be necessary, suturing the extensor tendon into the distal phalanx and using a wire pull-out technic.

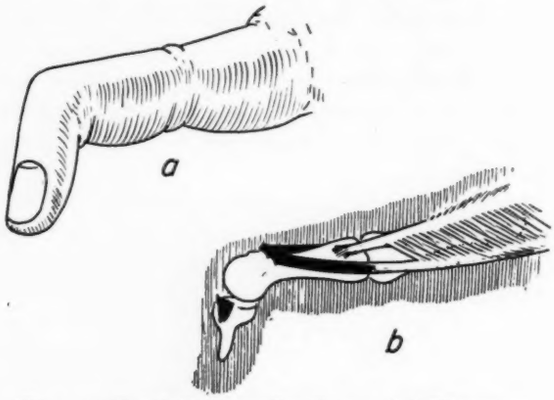


FIG. 12. Baseball or mallet finger. (*a*) Characteristic deformity; (*b*) rupture of extensor apparatus over distal interphalangeal joint.

Buttonhole Rupture of the Extensor Tendon. The extensor communis insertion into the middle phalanx may be torn from the middle phalanx permitting the lateral bands of the extensor mechanism to slide laterally over the interphalangeal joint, producing a characteristic deformity with the proximal interphalangeal joint being flexed and the distal interphalangeal joint extended. (Fig. 13.) Conservative therapy with immobilization in a plaster cast may be successful, but usually such a finger will require operation for reinsertion of the communis tendon into the middle phalanx.

Rupture of the Extensor Pollicis Longus Tendon. This tendon is long and thin and slides through a groove of the distal end of the radius beside Lister's tubercle. Any roughening of this groove due to an old Colles fracture or to arthritis will cause an eventual rupture of the tendon. The characteristic position is the thumb held in flexion with an inability to extend the distal interphalangeal joint. Trevor[35] recently reported nine cases of this disease—all occurring in women—with the interval between fracture and tendon rupture varying from four weeks to one year. He raises the question as to whether or not the rupture is due to a roughening of the tendon by trauma. He thinks that the most logical explanation is that the mesotendon tears at the time of the original injury thus interfering with the blood supply of the tendon, resulting in an avascular necrosis. Operative findings supported his theory, for the tendon appeared swollen, yellowish and bruised. The treament of this condition is surgical repair of the tendon whenever possible. Trevor did not find it necessary to approximate the ends, but occasionally a strong nylon suture threaded criss-cross through the tendon and across the tendon gap sufficed. A plaster cast is applied allowing a few degrees of flexion at the interphalangeal joint, and this is maintained for three weeks. An alternative procedure is suturing the extensor longus pollicis tendon to that of the extensor indicis proprius. In cases in which none of these methods are possible, the method of Furlong[36] in which a tenodesis between the distal ruptured tendon and the radius would give active extension of the terminal thumb joint on flexion of the wrist joint.

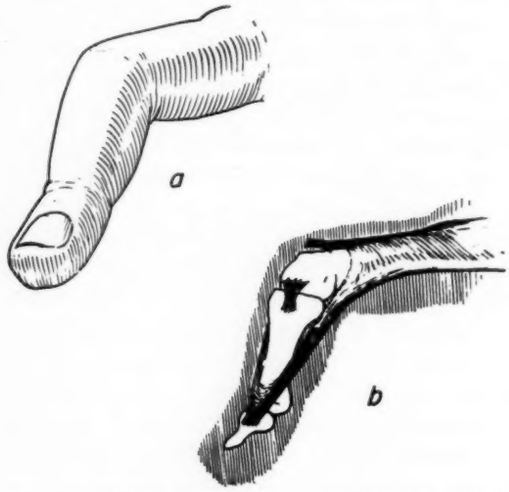


FIG. 13. Buttonhole rupture of extensor tendon. (*a*) Characteristic deformity; (*b*) extensor tendon torn from middle phalanx permitting lateral bands to slide over proximal interphalangeal joint.

Rupture of Extensor Communis Digitorum Tendon. Various slips of the extensor communis digitorum may rupture as they move across an irregularity in the radius due to an old fracture of the wrist. Gladstone[37] recently reported two such cases. Treatment of this is surgical repair of the involved tendon.

James[38] has recently reported pathologic ruptures where the tendons of the flexor

pollicis longus and the flexor profundus to the index finger were completely divided and appeared to be due to a fraying from continued motion across a semilunar bone affected with Kienböck's disease.

Tendon Grafts. A tendon graft will be necessary when tendons are destroyed by trauma, burns or infection, or when a primary repair has been unsuccessful. The most frequent site for such a tendon graft is the "danger zone" of the fingers. Such grafts are performed within four to six weeks from the time of injury or disappearance of infection. If the soft tissue is not satisfactory over the tendon site, some skin-grafting procedure will be necessary to supply skin and subcutaneous tissue. Physiotherapy to improve joint motion is necessary, since joint function must be as close to normal as possible before a graft will be successful. It is also essential that the moving part have a good nerve supply. If the nerves are damaged, they should be repaired at the time of the grafting if possible.

After the proper incisions are made in the hand to expose the diseased tendons, they are carefully dissected from the scar tissue. Care is taken to see that the annular ligaments in the fingers are preserved to prevent bowing of the tendon graft. In the fingers the flexor digitorum sublimis and profundus are both removed from their insertion to the distal third of the palm.

The source of the tendon graft is usually the palmaris longus tendon, when present, or the long extensor tendons of the toes. Some authors have suggested the use of the short extensor tendons of the toes because of their thinness. In the experience of the Boston City Hospital Hand Service, these usually are not of sufficient length to permit an approximation of the tendon in the proximal third of the palm, and for this reason their use is not justified. The donor tendon should be excised through longitudinal incisions, and as much paratenon as possible should be preserved.

The graft is threaded through the preserved annular ligaments or through the bed of the tendon sheath. Using a wire pull-out technic, the distal portion of the graft is inserted into the distal phalanx either through a hole or a bone flap. The proximal portion of the tendon is sutured to the proximal profundus tendon in the proximal third of the palm. The tension on the graft at the time of repair is an important factor. There should be just a slight amount of tension on the suture line with the wrist and finger slightly flexed. (Fig. 14.) If there is a great disparity in the tendon graft and the motor tendon, they may be sutured, as suggested by Allen,[39] with the thin graft inserted through a stab wound in the proximal motor tendon and sutured with fine silk sutures.

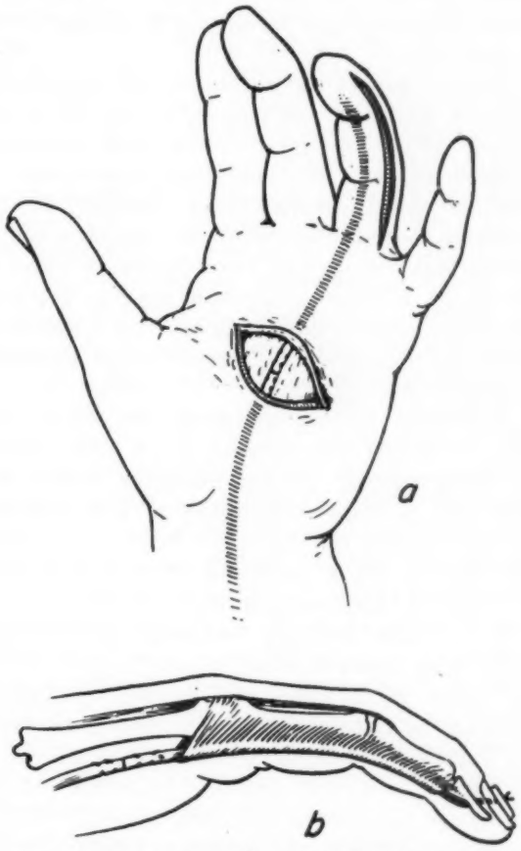


FIG. 14. Tendon grafting. (*a*) The donor graft extends from the distal phalanx of the finger to the proximal third of the palm. (*b*) The distal end of the graft is inserted into the distal phalanx, and the proximal end is sutured to the profundus tendon in the proximal third of the palm using a silk suture technic. The anastomosis line is thus placed outside the digital tunnel.

Flynn[40] notes that the tendon grafts may be inserted into the distal phalanx using a plain catgut pull-out suture with the ends of the catgut being tied over a button on the dorsum of the distal phalanx. He uses catgut in this location, for it is well known that catgut increases adhesion formation, and he believes that this is desirable at this point.

If the tendon injury involves the flexor profundus tendon distal to the proximal interphalangeal joint with normal flexor digitorum

sublimis function, a grafting procedure is probably not indicated but some form of arthrodesis with the distal joint at 20 degrees flexion should be performed.

When the flexor pollicis longus tendon has to be replaced, tendon grafting may be done. The procedure of transferring the flexor digitorum sublimis of the ring finger to replace the flexor pollicis longus tendon in the thumb may be followed.

Tendon grafts for extensor tendon injuries are not indicated as frequently as flexor injuries. When necessary, the long extensor tendons of the third and fourth toes can be used as donor grafts, or a transfer of the extensor indicis proprius or extensor digiti quinti may be useful. The correct position of the hand following extensor tendon grafting is 5 to 15 degrees dorsiflexion of the wrist with about 5 degrees flexion in the metacarpophalangeal joints.

Following tendon grafting the hands are kept in the proper position by a plaster cast for three to four weeks, following which the cast and sutures are removed and physiotherapy instituted. The patient's mental drive and iniative are of profound importance in the recovery of function in tendon grafts.

Boyes[41] has recently analyzed 138 tendon grafts with flexor tendon damage in the "critical" zone. When there was no joint damage or scarring, he obtained good results. In all cases the fingers could be flexed within 1½ inches of the palm; in 90 per cent of the cases, within 1 inch; in 35 per cent, ½ inch; in 10 per cent, complete flexion to the proximal digital crease. When there was joint damage, scarring or multiple damage of the fingers, however, the results were not as good. The author obtained best results with stainless wire sutures which were used at the insertion in the phalanx and also as the suture in the palm together with deliberate excision of all remaining sheath tissue. The best donor tendon was the palmaris longus with its entire paratenon.

Stenosing Tendovaginitis. This is an inflammatory condition of the tendon sheath in which the sheath becomes constricted and the underlying tendon becomes inflamed, developing adhesions with subsequent limitation of function of the tendon. It appears wherever a tendon glides under a fibrous tunnel such as the annular ligaments over the metacarpal heads of the fingers and thumb, and the fibrous sheaths over the radial styloid under which the tendons of the abductor longus pollicis and extensor pollicis brevis glide.

Lapidus and Fenton[42] have recently reviewed 369 cases in which 420 tendon sheaths were involved. The sheaths of the abductor longus pollicis and extensor pollicis brevis made up 39 per cent of all cases; the flexor pollicis longus, 31.9 per cent; the flexor sheath of the third finger, 10 per cent; the flexor sheath of the fourth finger, 10.9 per cent with occasional involvement of the flexor sheath of the index finger, fifth finger and other extensor tendons of the wrist.

Burman[43] has reported a group of cases of this disease which were confined to extensor tendons of the wrist other than the abductor pollicis longus and the extensor pollicis brevis.

In every case reported by Lapidus some occupational motion was present which seemed to account for the location of the disease. He found that the condition was more prone to develop in "a new worker unaccustomed to the job, in an old worker out of training and returning to his task after a period of idleness, or in a worker who is asked to speed up his output to meet a new production quota."[42] As further evidence that trauma may produce this condition, Engel[44] reported three cases which appeared to be produced by exposure to high temperature. It is obvious that all such cases are not occupational, since Sprecher[45] has recently reported a series of twelve cases appearing in patients before the age of three.

When the disease occurs in the fibrous sheaths of the fingers (trigger finger), there is at first difficulty in movement of the fingers associated with pain on flexion. As the disease progresses, a snapping may be felt and heard as the finger is extended and flexed. If the disease is allowed to continue, it may become locked in flexion or extension. Associated with this there will be tenderness over the metacarpal head of the involved finger at the site of the disease process, and a bulbous enlargement of the tendon may be felt in the same location. Treatment is incision of the constricted area of the tendon sheath.

Stenosing tendovaginitis of the tendon sheaths of the abductor pollicis longus and the extensor pollicis brevis was first recognized by de Quervain in 1895. Since that time many articles have been written about this subject. This occurs much more frequently in women.

In Lapidus' series, 75 per cent of 369 patients were female.[42] The disease is manifested by pain and tenderness over the styloid process of the radius. Motion of the thumb produces pain particularly on flexion. When the thumb is held flexed within the fist and the hand ulnar deviated, there is marked pain at the involved site.[46] There may be swelling over the involved tendons and occasionally erythema is present. Snapping or locking is never seen in this disease. Treatment may be conservative with immobilization in a plaster of paris cast. Surgical treatment, however, is simple and successful. Through a small transverse incision over the abductor pollicis longus tendon, the involved tendon sheath is incised longitudinally throughout the entire distance of the constriction. The presence of accessory tendons in the tendon sheath of the abductor pollicis longus is common. It is not necessary to excise these for relief of symptoms. Following surgery a pressure dressing is applied and active motion is begun shortly after operation.

Cohen[47] reported several cases in which recurrence of the disease followed surgery. On reoperation it was found that the tendon of the extensor pollicis brevis passed through a separate fibrous canal which was surrounded by a typically thickened synovial sheath, the resection of which gave relief of symptoms.

Murphy[48] similarly reported two cases of Quervain's disease in which the constriction involved only the extensor pollicis brevis which was in a separate compartment from the abductor pollicis longus.

Howard, Bunnell and Pratt[49] have recently reported on the treatment of Quervain's disease with hydrocortone. Several cubic centimeters of this were injected into the involved tendon sheath, with relief of symptoms.

Traumatic Tendinitis. Occasionally because of prolonged use, inflammation of a tendon may occur which may persist for weeks or months. It is most frequently seen in the tendons near the insertions of the extensor carpi radialis longus, in which case there will be tenderness over the involved tendon and crepitation can be felt on flexing and extending the wrist.

Burman[50] has reported on several cases of tendinitis at the insertion of the common extensor tendons of the fingers. One of these fingers was explored, and friable granulation-like tissue was found beneath the tendon. Histologic section showed vascularized fibrotic tissue.

Seidenstein[51] has reported numerous cases of a similar disease occurring along the course of the flexor carpi ulnaris at or near the pisiform bone and along the course of the flexor carpi radialis at or about the greater multangular. Associated with the tenderness and pain in these areas, x-rays demonstrated calcific deposits in the peritendinous soft tissues.

Phalen[52] has similarly reported on calcium deposits around the tendon of the flexor carpi ulnaris associated with pain and tenderness over the involved area. The disease process is self-limited and usually responds to immobilization in a plaster of paris cast.

NERVE INJURIES

Healing of Nerve. When a nerve is injured, there are several microscopic and macroscopic changes which occur. The distal portion of nerve undergoes wallerian degeneration in which the axis cylinder disintegrates, the medullary sheath fragments, and the sheath of Schwann cells degenerate. Similar changes occur in the proximal nerve up to the first node of Ranvier. The cellular changes in the cell nucleus in the spinal cord occur during this same period. The axis cylinders begin to proliferate and emerge from the cut proximal nerve end where they become emeshed with an overgrowth of connective tissue from the perineurium, endoneurium, epineurium and sheath of Schwann. A recognized nodule thus appears at the end of the proximal nerve which has been called an "amputation neuroma." At the distal end of the nerve there may be some slight tendency for the connective tissue to grow and form a "distal glioma." If the freshly incised nerve is sutured or if in an old injury the scarred portions are cut away and the ends approximated, normal nerve healing may occur. The neurofibroblasts proliferate and bridge the gap between the nerve ends and will accompany and ensheath the regenerating nerve fibers down the distal nerve.[53] The Schwann cells play only a small part in the regeneration of nerve. The nerve fibrils continue to grow down the distal nerve essentially in the meshes between the cells of the degenerating distal nerve.[53]

The first clinical manifestation of regenerating nerve fibrils down a nerve are the occurrence of paresthesias along the nerve on per-

cussion. If the nerve is percussed, beginning distally and proceeding proximally, a point will be reached at which the patient feels a "tingling or buzzing" sensation with radiation of sensation down to the involved hand area. The advancing edge of this sensitive area in the nerve should be measured at intervals, using some bony prominence as a guide. This is known as Tinel's sign and is a rough test of nerve recovery, since one can never be sure that the nerve fibrils producing the paresthesias will function clinically. The clinical manifestations of nerve recovery are return of sympathetic tone, sensation and motor function. Various electrical technics have been used for testing of nerve recovery, such as electrodiagnosis and electromyography.[54] These are of great prognostic value since they may save many months of futile waiting for clinical manifestations in cases in which reoperation on a nerve or a tendon transfer is being contemplated.

Nerve Injury Patterns. Ulnar nerve: The area of sensory loss with ulnar nerve palsy consists of the volar and dorsal aspects of the palm and fingers on the ulnar side of the hand from a line bisecting the ring finger. If the injury occurs above the elbow, the flexor carpi ulnaris and flexor digitorum profundus to the fourth and fifth fingers are paralyzed. The intrinsic muscles of the hand innervated by the ulnar nerve are the abductor digiti quinti, flexor digiti quinti, opponens digiti quinti, the dorsal and volar interossei, the adductor pollicis, the deep head of the flexor pollicis brevis, and the lumbrical muscles to the fourth and fifth fingers. The interossei can be tested by abducting and adducting the fingers with the metacarpophalangeal joints held extended. It is well to remember that false abduction and adduction can occur through flexion and extension of the fingers. The abductor digiti quinti and the first dorsal interosseus are convenient muscles for testing, since the atrophy of these muscles is quite apparent in an injured hand. The adductor pollicis may be tested by requesting the patient to grasp a newspaper with both hands, pulling the hands away from each other without releasing the grip. When the adductor pollicis is paralyzed, the thumb is flexed at the interphalangeal joint because of contraction of the flexor pollicis longus (Froment's sign). If the palsy continues, trophic changes will take place in the fifth finger so that it will appear tapered, and nail and hair growth will change. The characteristic deformity of this injury is flexion of the fourth and fifth fingers with the thumb held in slight abduction. The flexion contracture of the fourth and fifth fingers is worse if the injury is below the innervation of the flexor digitorum profundus.

It is well to remember that anomalous innervation of the intrinsic muscles of the hand may take place. In an excellent study of this subject by Rowntree,[55] the previously mentioned so-called normal innervation of the intrinsic muscles was present in only 15.5 per cent of 226 cases. In 32 per cent of the cases the flexor brevis pollicis was solely supplied by the ulnar nerve; in 33 per cent both heads were supplied completely by the median nerve. Other outstanding anomalies were as follows: All the thenar muscles were supplied by the ulnar nerve in 2 per cent of cases; all the thenar and the adductor pollicis muscles were supplied by the median nerve in 2 per cent of cases; all the thenar muscles, including the adductor pollicis and the first dorsal interosseus, were supplied by the median nerve in 1 per cent of cases.

In a study of peripheral nerve injuries during World War II Murphey et al.[56] found anomalous innervation in some of the intrinsic muscles of the hand. In four cases of 698 ulnar nerve injuries, the first dorsal interosseus was found to be innervated by the median nerve, either by a branch given from the median nerve to the ulnar nerve in the forearm or by way of a branch directly to the first dorsal interosseus muscles in the hand. In two cases the flexor pollicis brevis had an anomalous innervation; in one case the abductor minimi digiti was supplied by the median nerve; in another case the opponens pollicis was supplied by the ulnar nerve.

Prognosis following repair of ulnar nerve injuries is excellent as far as return of sensation and motor function in the large forearm muscles are concerned. However, the intrinsic muscles of the hand are so small that they usually atrophy before they are renervated. Cases of ulnar nerve injury with the best prognosis as far as intrinsic motor function is concerned are those in and about the wrist in the younger age group.

Median nerve: The sensory loss in median nerve palsy consists of anesthesia on the volar aspect of the palm and fingers lateral to a line bisecting the ring finger. The dorsum of the distal phalanx of the thumb, index, middle and

radial half of the distal phalanx of the fourth finger are similarly involved. The median nerve supplies the following muscles of the forearm: pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum sublimis, flexor digitorum profundus to the second and third fingers, flexor pollicis longus and pronator quadratus. The intrinsic muscles in the hand supplied by the median nerve are the abductor pollicis brevis, the opponens pollicis and the superficial head of the flexor pollicis brevis. The possibility of anomalous innervation as mentioned in the previous section must be considered in certain cases. Trophic changes develop in the thumb, index and middle fingers consisting of loss of soft tissues, tapering of the fingers, stiffening of the joints and changes in the hair and nail growth. The characteristic deformity is loss of substance of the thenar muscle group together with an adduction contracture of the thumb.

The prognosis following repair of the median nerve is excellent for sensation and large muscle function. The intrinsic muscles of the thenar eminence, however, rarely recover unless the nerve injury is in the region of the wrist in the younger age group. Causalgia, which occurs in about 5 per cent of peripheral nerve injuries, is most commonly seen after median nerve injuries.[57]

Combined median and ulnar nerve palsies: The characteristic deformity of this condition is flexion of the interphalangeal joints of the fingers with extension or hyperextension of the metacarpophalangeal joints. This is a so-called "clawhand," or as described by Bunnell,[58] "the intrinsic minus position."

Radial nerve: The sensory loss in radial nerve palsy is on the dorsolateral surface of the hand. In the forearm it supplies the following muscles: brachioradialis, extensor carpi radialis longus, extensor carpi radialis brevis, extensor carpi ulnaris, extensor communis digitorum. The deep branch of the radial nerve supplies the supinator muscle, extensor indicis proprius, extensor pollicis longus, extensor pollicis brevis and abductor pollicis longus. The obvious deformity with the complete radial nerve palsy is the wrist, finger and thumb drop. When the deep branch of the radial nerve is severed, there is a characteristic inability to extend the thumb which often may be overlooked. In particular, it should be watched for in fractures of the head of the radius. The prognosis following repair of the radial nerve is excellent, since most of the muscle bellies supplied by this nerve are large. During World War II numerous repairs were attempted of the deep branch of the radial nerve with considerable success. Mayer and Mayfield[59] studied fifty-eight cases, in which end-to-end suture was possible in thirty-nine. A follow-up study of twenty-six of the patients who were seen more than six months after surgery revealed that 84 per cent showed partial or full recovery following the procedure.

Methods of Repair. Nerve ends are approximated using No. oooooo silk sutures which pass through the epineurium of the nerve. The suture should not be allowed to extend into the nerve for fear of damage to nerve fibrils with subsequent poor recovery. In old injuries the amputation neuroma should be excised back to normal nerve fibrils, and the distal end should be freshened to relatively normal tissue. The nerve approximation is done similarly to a fresh injury.

Since it is a primary requisite to suture the nerve without tension, there are several ways by which this can be done. Simple neurolysis of the involved nerve over a considerable portion of its normal bed will often serve the purpose. Joints may be flexed on either side of the nerve repair in order to gain distance. In the case of the ulnar nerve, it may be taken from its normal bed behind the medial epicondyle and transplanted anteriorly, taking care not to damage the nerve fibers going to the flexor carpi ulnaris and flexor digitorum profundus. An excellent account of the technic for transplanting the ulnar nerve has been presented by Learmonth.[60] The radial nerve may be translocated anterior to the humerus in order to achieve a similar purpose. In rare instances with extensive loss of nerve tissue it has been necessary to shorten bone length in order to anastomose nerves.[61]

Aftercare. Following operation the involved extremity should be immobilized in a plaster of paris cast in a position consistent with the least tension on the suture line for a period of three weeks. After removal of the cast and sutures, subsequent care is devoted to physiotherapy to maintain joint function of all the joints in the involved extremity and adequate splinting to prevent overstretching of paralyzed muscles. For radial nerve injuries a cock-up type of splint, preferably a dynamic one, is

valuable. For median nerves a splint to restore opposition to the thumb and to maintain the web space between the thumb and index finger should be utilized. The usual ulnar nerve paralysis requires no particular splinting. With the combined median and ulnar nerve palsies, however, some form of "knuckle duster" is indicated which will keep the metacarpophalangeal joints in a position of about 20 degrees flexion together with an opponens splint.

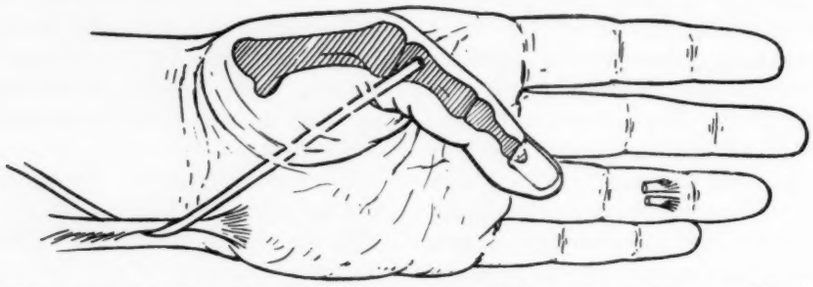


FIG. 15. Tendon transfer for opponens palsy. The flexor digitorum sublimis tendon to the ring finger is rerouted through the wrist, looped around the flexor carpi ulnaris muscle as a pulley and brought subcutaneously to the proximal phalanx of the thumb according to the principle of Bunnell.

Tendon Transfer. When recovery of motor function does not occur following nerve suture, various tendon transplants may be done to restore function. In nerve injuries where motor return is not expected for twelve to eighteen months there is an increasing tendency to perform the tendon transfers early, using them as a type of dynamic splinting.

The wrist, finger and thumb drop of radial palsy may be successfully treated by some modification of the Jones tendon transfer.[62] An excellent presentation of this subject was recently written by Scuderi[63] who described his experiences in World War II. He believed that the best results were obtained by transferring the flexor carpi ulnaris into the extensor tendons of the second, third, fourth and fifth fingers; and the palmaris longus into the severed extensor pollicis longus tendon. If the palmaris longus tendon is absent, the flexor carpi radialis should be inserted into one of the thumb tendons. Whenever possible it is well to keep one of the flexors of the wrist joint intact, for if all three are used for the transfer there is a great weakness of wrist flexion. The author has seen one case in which the wrist became fixed in extension because of this. It is well to maintain active wrist flexion, since in many cases the beneficial results obtained from this tendon transfer is due to the tenodesis whereby flexion of the wrist produces extension of the fingers.

Restoration of opposition and abduction of the thumb following nerve injury is of primary importance. The principles outlined by Bunnell[64] in this regard are still of paramount importance. He stated that the tendon from its insertion in the thumb should pass subcutaneously in the direction of the pisiform bone; the insertion of the tendon should be on the dorsal ulnar aspect of the base of the proximal phalanx of the thumb and should pass directly over the summit of the metacarpophalangeal joint, not distal to it. Various combinations of pulleys, tendons and motors have been used for this operation, using the foregoing principles. One of the most common variants of this operation is the one in which the flexor digitorum sublimis to the ring finger is removed from its attachment in the middle phalanx, pulled out through the wrist, looped around the flexor carpi ulnaris and brought subcutaneously through a tunnel to be inserted into the proximal phalanx of the thumb. (Fig. 15.)

A review of seventy-five cases in which various modifications of the Bunnell procedure were utilized was presented by Kirklin and Thomas.[65] Goldner and Erwin[66] have analyzed various procedures of value for paralysis of the thumb. If some of the extrinsic strength of the thumb is missing, it must be restored before the opponens operation is done. For instance, if the extensor pollicis longus is weak, the extensor carpi radialis longus can be sutured to it. If the flexor pollicis is weak, the flexor digitorum sublimis to the index or middle finger can be sutured into the tendon at the wrist. If there is a severe adduction contracture of the thumb, it will have to be overcome by

various procedures such as capsulotomy of the metacarpophalangeal joint of the thumb, cutting of the fascia between the first and second metacarpals, stripping the first dorsal interosseus from the first metacarpal bone, division of a contracted adductor pollicis muscle at its point of insertion and capsulotomy of the carpometacarpal joint. After the thumb is mobilized in a plaster cast for three weeks, a sublimis transplant can be done to restore opposition.

Thompson[67] has described another operation for opponens paralysis in which the flexor digitorum sublimis tendon is removed through an incision in the palm after sectioning it at the base of the ring finger. Another incision is made on the dorsal surface of the proximal phalanx of the thumb, and the flexor sublimis tendon is brought through a subcutaneous tunnel across the thenar eminence. The tendon is split longitudinally, and one end is inserted into the distal end of the metacarpal of the thumb and the other into the proximal phalanx.

It does not seem that this operation restores abduction of the thumb as well as the Bunnell procedure. Kirklin and Thomas[65] mention an excellent point about using the Thompson operation whenever there is scarring over the distal half of the forearm which would prevent a tendon transfer in this area.

Whenever it is impossible to do a tendon transfer to restore opposition of the thumb, a bone procedure may be necessary such as a metacarpal osteotomy or a bone wedge between the first and second metacarpal. It would seem that for a bone block to be successful one should have good flexion of the fingers together with strong abduction of the index finger.

Brooks[68] has recently reviewed sixteen cases of intermetacarpal bone grafts for thenar paralysis, and there were only two complete failures in the entire group. It is interesting that five grafts failed to fuse at one end and it did not seem to affect function very much. As a matter of fact, in one patient pseudoarthrosis developed which seemed to help the function of the thumb. He mentioned that if one was in doubt about the benefit which could be derived from such an operation, a trial plaster fixation of the thumb in the position of function might be indicated. Smillie[69] uses a Kirschner wire which transfixes the bone block lengthwise as well as impaling the metacarpals, thus fixing this fusion without the use of a plaster of paris cast. This pin is left in the bone for eight weeks.

Paralysis of the interosseus and lumbrical muscles results in the "clawhand or intrinsic minus hand." There is a flexion contracture of the interphalangeal joints, and the metacarpophalangeal joints are held in extension or hyperextension. Extension of the middle and distal phalanges of the fingers can be performed by the extensor communis tendon through its action on the lateral bands if the proximal phalanges are held in slight flexion or if the wrist is flexed. On the other hand, when these metacarpophalangeal joints and even the wrist are held in extension, the extensor tendons have little affect on extension of the distal phalanges. If contraction of the metacarpophalangeal joint takes place so that hyperextension is impossible, the claw deformity is less obvious. This is also true if the nerve lesions responsible for the claw deformity are high enough to paralyze the flexors of the fingers. For most cases of clawhand, the Bunnell procedure has proven of great benefit. In this operation the flexor digitorum sublimis tendons are removed from their insertions in the middle phalanx, withdrawn from the palm, split and passed through the lumbrical canals to be attached to the transverse fibers and lateral bands of the fingers. The split strands from each tendon should go either to the radial or ulnar side of the fingers, so that lateral motion of the fingers will be possible. Each finger should have at least one tendon attached to it, so that the distal two joints can extend and the metacarpophalangeal joint flex.[23] If sufficient tendons are not available, the extensor proprius tendon of the index finger can be utilized to restore abduction to the index finger as well as extension of the distal joints. The hands and fingers are splinted for three weeks with the wrist in flexion. On the fourth week light motion may be begun, with physiotherapy following this.

Brand[70] has modified this operation slightly in restoring function to hands paralyzed by leprosy. An incision is made on one side of the finger only, and the tendon transplants are inserted to the radial side of each finger. He uses the index finger tendon for the radial side of the index finger. The long finger tendon is split and one-half is used for the long finger and one-half for the ring finger. The fifth finger tendon is used only for the fifth finger, thus

saving the ring finger sublimis for use in restoring opposition to the thumb. As each tendon is brought through the lumbrical canal, a slit is made in the lumbrical tendon. The transferred tendon is passed through this aperture and sutured to the dorsal edge of the extensor carpi ulnaris is split from one-third the distance above its insertion and left attached to the metacarpals. Each of these strands is split in two, and they are routed through the interosseus space to the radial side of each finger anterior to the transverse metacarpal ligament.

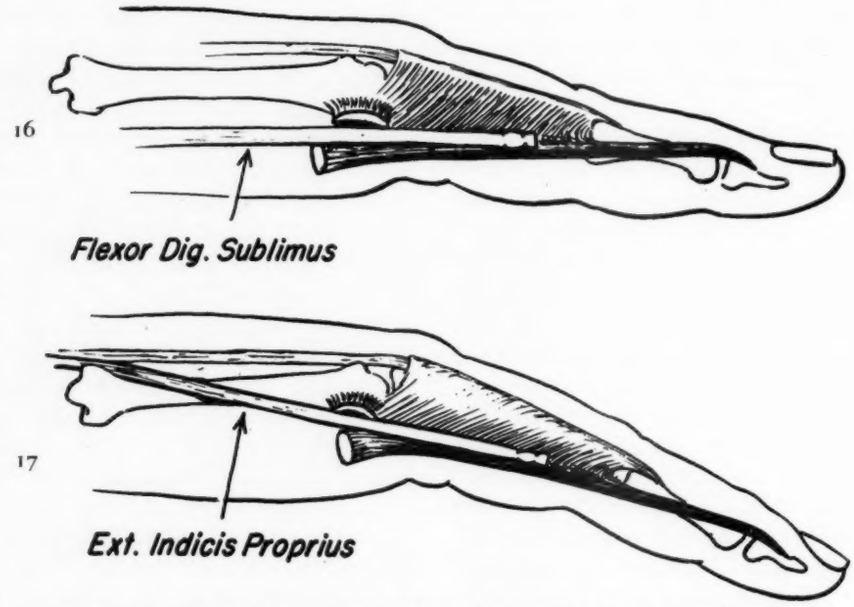


FIG. 16. The Bunnell procedure for paralysis of interosseus and lumbrical muscles. Slip of flexor digitorum sublimis is removed from finger and passed through lumbrical canal to insert into lateral bands.

FIG. 17. Fowler procedure for paralysis of interosseus and lumbrical muscles. Slip of extensor indicis proprius is threaded between metacarpals and carried anterior to transverse metacarpal ligament and inserted into lateral bands of extensor apparatus.

expansion just proximal to the interphalangeal joint.

Riordan has recently reported on the use of a tenodesis operation in similar cases based on the so-called Fowler principle, i.e., if the metacarpophalangeal joint is stabilized in a position of slight flexion the extensor communis tendon will be able to extend the distal joints. He used the Fowler procedure in which the extensor indicis proprius and the extensor digiti quinti proprius are split, each into two strands, and each slip is then passed through the interosseus space anterior to the transverse metacarpal ligament and inserted into the extensor aponeurosis. Each slip is inserted into the radial side of each phalanx.

In cases in which there is not sufficient strength in the extensor muscles Riordan[71] has devised his own procedure in which one-half of the extensor carpi radialis and extensor

The hand is mobilized in a pressure dressing with the wrist in dorsiflexion, the metacarpophalangeal joints in flexion and the distal phalanges in extension for three weeks. Following this the cast is removed and active motion begun. This operation would appear to be most successful when there is marked hyperextension of the metacarpophalangeal joint. Figure 16 demonstrates the principles involved in the Bunnell procedure for interosseus and lumbrical muscle atrophy. Figure 17 demonstrates the principles involved in the Fowler procedure. In cases in which none of these procedures are practical some form of arthrodesis of the finger joints will be necessary.

Compression Palsy. The *median nerve* may be compressed in the carpal tunnel, producing a typical median palsy in the hand. There is usually pain over the median nerve distribution. As the disease progresses, there will be a

definite pattern of hypesthesia or anesthesia over the median nerve distribution and opposition of the thumb may disappear. Percussion of the median nerve at the flexion crease of the wrist will produce paresthesias along the median nerve.[72]

The cause of this condition is varied. Occasionally some disturbance in the carpal canal such as a fracture or dislocation might so narrow the tunnel as to produce the syndrome. Acute compression by hemorrhage has occasionally been the cause. However, in most cases it appears to be due to occupational strain. In extension of the wrist the pressure in the carpal tunnel is three times greater than when the wrist is flexed.[73] Also when the wrist is flexed, the edge of the transverse carpal ligament can pinch the median nerve. It is obvious that any action involving extension and flexion of the wrist could conceivably lead to this condition. The author has recently seen a case in an elevator operator in which, at the time of operation, the flexor tendons appeared slightly hyperemic and thickened as if they were the site of a chronic tendinitis. Their increased bulk may have helped produce the compression palsy.

Treatment of this condition is relatively simple and involves incision of the transverse carpal ligament which is exposed through an S-shaped incision across the wrist. The ligament should be sectioned along its medial border to minimize the possibility of damage to the motor branch of the median nerve. It is essential that the diagnosis in this case be made before opponens palsy occurs, since this may never return.

Reports of *tardy ulnar palsy* are increasing in the surgical literature. It is usually seen as a complication of a fracture of the lateral humeral condyle resulting in valgus deformity of the elbow. If the elbow is flexed, the nerve becomes stretched and may even slip over the edge of the condyle. There is usually paresthesia and hypesthesia over the ulnar nerve distribution followed by increasing weakness of the intrinsic muscles. A Tinel sign will be elicited over the ulnar nerve in the condylar tunnel, and a tender enlargement of the nerve may be found in this area.

McGowan[74] has presented a series of forty-six cases of this disease. In this series twenty-one cases were due to an old fracture; four were due to osteoarthritis with no history of fracture; four presented bony irregularities in the ulnar groove; three were caused by recurrent dislocation of the ulnar nerve; two were due to congenital valgus deformity with an anterior dislocation at the head of the radius; three were due to an old soft tissue injury at the site of the ulnar nerve with subsequent compression by scar tissue. Three of the cases presented no mechanical abnormality of the elbow joint. The occupation of two of these patients required much hammering. Another case was that of a student who had the habit of sitting for many hours with his head on the left hand and the elbow pressing against the table.

Treatment of this condition consists of translocation of the ulnar nerve anterior to the epicondyle. A good account of this operation has been previously mentioned.[60] As with tardy median nerve palsy, it is essential to operate before there is definite wasting of the intrinsic muscles. This is well emphasized in McGowan's report in which all the cases improved as far as sensation was concerned, but when there was wasting of the interosseus muscle with no voluntary power there was little motor improvement.

Brooks[75] has recently reported a series of cases involving nerve compression by ganglia. Four cases were ganglia near the elbow causing compression of the ulnar nerve. The ganglia were attached to the capsule of the elbow joint and compressed the nerve. There were four cases of ganglia at the wrist, two causing an ulnar nerve compression and two producing a median nerve compression.

The *radial nerve* may be compressed in the healing of a fractured humerus resulting in late radial nerve palsy which may require operation on the fracture site with neurolysis of the nerve.

AMPUTATIONS AND SEVERE LACERATIONS

In digit amputations, especially of the thumb and index finger, every effort should be made to maintain the length of the finger. If bone is not exposed at the tip, the wound may be covered with a split-thickness skin graft. If bone is exposed, either a full-thickness skin graft or a small flap raised from the thenar eminence should be performed. With the full-thickness graft it is often difficult to obtain pressure on the tip of the finger. This may be accomplished by leaving the skin graft sutures long and tying them over a piece of foam rubber. (Fig. 18.)

Kutler[76] has described a procedure for fingertip amputation in which a V-shaped flap is incised on either side of the finger but left attached to the underlying subcutaneous tissue. These flaps are brought together in the midline and the resultant lateral defect closed by the usual V-Y procedure.

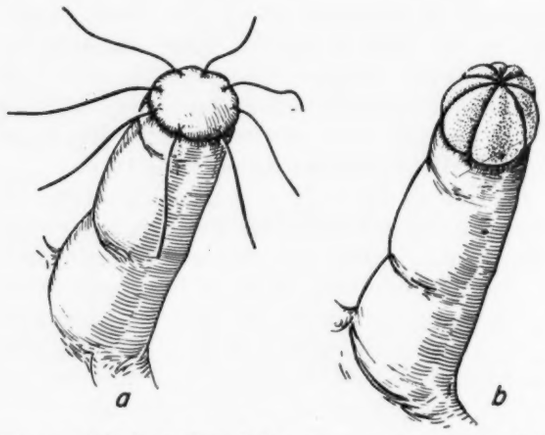


FIG. 18. Method of applying pressure to fingertip skin grafts. (*a*) Skin graft sutures are left long. (*b*) They are tied over a piece of foam rubber.

Since fingertip injuries may be painful because of underlying amputation neuromas, a careful search should be made for the digital nerve at the time of each amputation. Sufficient nerve should then be excised so that it will retract from the fingertip.

It must be remembered that grafting of fingertips prolongs the healing time of the injury. In many cases in which the patient is elderly, shortening of the finger by plastic amputation, leaving the scar on the dorsum of the finger with a long volar flap, may give quicker wound healing.

When an entire finger needs amputating, one can occasionally close the defect in an adjacent portion of the hand by fileting the finger and covering the defect with the salvaged skin. Defects on the volar surface of fingers, where tendons are exposed, can be closed by swinging a skin flap from the lateral side of the finger and covering the lateral defect with a split-thickness skin graft.

On other areas of the hand various combinations of split-thickness skin grafting plus shifting of skin flaps will be necessary to cover serious wounds. It is essential that the wound be covered completely to prevent subsequent scar tissue formation in the hand. A review of skin grafting for hand coverage has been written by Dubitoir.[77]

BURNS

Early treatment of the burned hand consists of minimal débridement and a firm pressure dressing. The hand should be immobilized in a position of function, care being taken that the gauze dressing extends between the fingers to prevent their adherence to each other. The fingers should be spread apart with all the joints placed in flexion, and the thumb brought into a position of opposition and abduction. The tips of the fingers should be exposed so that the state of circulation in the hand can be observed. After several days the dressing may be changed daily and the hand soaked in peroxide or half-strength Dakin solutions. Clean granulating areas should be skin grafted as soon as possible with split-thickness skin grafts of approximately $^{12}/_{1000}$ inch in thickness. As soon as possible after the grafting procedures, motion should be started in the fingers. Subsequent keloids or thin constricting scar areas should be excised and skin grafted with a split-thickness skin graft.

Burns on the dorsum of the hand are more frequent than others and much has been written concerning them.[78–80] When the burn does not involve the underlying tendons, the granulating burn area or thin atrophic scar area should be excised completely in a bloodless field, taking care to avoid the underlying dorsal veins and nerves. Excision should be so planned that the resultant scar will not cross a flexion crease at the wrist or finger joints. A split-thickness skin graft is obtained and tailored to fit the defect and sutured in place. The web space of the fingers demand attention, since often adduction contractures may take place in this area. The skin graft should completely cover the web space down to the volar surface, taking care that the digital nerves in this area are not damaged. When the burns are deep and underlying tendons or bones are destroyed, a split-thickness graft is not adequate and a pedicle flap of skin and subcutaneous tissue must be used followed by subsequent reconstructive procedures. Even when the dorsum has been properly skin grafted, contractures of joints and connective tissue in the hand may remain which must be corrected by proper dynamic splinting and physiotherapy.

Burns on the volar surface of the palm of the

hand may be treated with split-thickness skin grafts if the underlying tendon sheaths are preserved. If they are destroyed, a pedicle flap will be necessary to permit subsequent reconstructive procedures.

Burns on the fingers may usually be adequately covered with split-thickness skin grafts. As a rule, if the burn is so severe as to destroy underlying tendons or joints, reconstructive procedures in the fingers are impossible.

The foregoing methods of burn treatment often result in much fibrosis due to the skin grafting of granulation tissue. There is an increasing tendency to excise these burns at the time of injury with immediate split-thickness skin grafting.

BONES

A few general principles should be emphasized. Finger fractures should be immobilized in a position of 15 degrees flexion at each joint. Only the injured finger should be immobilized to prevent limitation of motion in other fingers. Watson-Jones[81] suggests that even this is not enough, but that every effort should be made to exercise actively the uninjured fingers through the complete range of motion. Immobilization is seldom necessary beyond three weeks. Beware of limitation of motion of the shoulder and insist on putting the shoulder through a full range of motion every day!

Fractures of the Distal Phalanx. These fractures are often associated with a subungual hematoma which may have to be evacuated to prevent pain and possible secondary infection. This is easily done by turning up a flap of nail using a scalpel. Most terminal phalanx fractures heal without difficulty and, as a rule, can be treated as soft tissue injuries. If there is an avulsion of the extensor tendon from the distal phalanx with a resultant "baseball finger," it should be treated as discussed in the tendon section. The hyperextension required in treating these injuries, however, should be modified in cases in which the distal interphalangeal joint is involved either as a chip fracture or as a dislocation. These will usually result in a stiff joint and should be immobilized with the distal interphalangeal joint in a slight degree of flexion.

Middle and Proximal Phalanges. These are always treated in flexion, and position can be maintained by a plaster of paris trough. If there is overriding of the fracture ends with bowing, it may be necessary to use traction. This may be accomplished by means of a coat hanger which is incorporated in a plaster gauntlet. (Fig. 19.) To prevent the finger from slipping between the wire struts, adhesive tape or plaster of paris slabs are used. Pulp traction

FIG. 19. Method of applying traction to fingertip. A coat hanger is incorporated in a plaster of paris cast and curved so that traction is applied with the finger in the position of function. Upper illustration demonstrates traction wire placed through periosteum of distal phalanx of finger.

through the terminal phalanx is usually adequate providing the traction wire is placed through the periosteum of the distal phalanx. Adhesive traction may occasionally be used but must be watched carefully for fear of slipping or skin necrosis. When traction is applied, it is important to remember that the fingers in the flexed position do not lie parallel to each other but instead point approximately to the scaphoid tubercle. (Fig. 20.) Frequent x-ray checks of the immobilized fracture should be performed to be sure that anterior bowing of the fracture site is not taking place, since this may interfere with function of the flexor tendons. It is well to emphasize that if a fracture goes into one of the joints, the prognosis is poor since there will be much limitation of motion in the involved joint. Total immobilization should not be more than three weeks. At the end of this period active motion should be instituted, and some form of protective splint may be necessary to prevent displacement of the fracture site.

Metacarpal Fractures. Fractures of the base seldom present a problem and may be immobilized for three to four weeks in a dorsal

splint, being sure to keep the fingers and thumb moving.

Most fractures of the shaft of the metacarpals can be treated by manipulation and immobilized in a plaster gauntlet, keeping the fingers and thumb free and moving. If the

FIG. 20. Flexed fingers point essentially to scaphoid.

fragments cannot be held without serious bowing and overriding, open reduction is probably indicated. Intramedullary pinning with a Kirschner wire offers a satisfactory means of immobilization.[82]

Fractures of the neck are the most common fractures found in the metacarpal bones. If there is only slight displacement of the head, the hand may be splinted in this position. However, any serious displacement of the head requires manipulation. This is easily done by the maneuver described by Jahss,[83] where the flexed metacarpal phalangeal joint is pressed backward in the long axis of the phalanx. When the reduction is accomplished, the metacarpophalangeal joint may be immobilized by a plaster cast, taking care that felt is placed over the extensor surface of the flexed finger and in the space between the finger and palm. This fracture is immobilized for three weeks and followed by gradual active motion. Kirschner wire fixation occasionally may be used to immobilize this fracture.

Thumb Fractures. Fractures of the neck, shaft and extra-articular base usually require reduction and immobilization in a plaster gauntlet with the thumb extended and abducted.

Bennett's fracture is an intra-articular fracture dislocation of the thumb at the carpometacarpal joint, presenting a triangular frag-

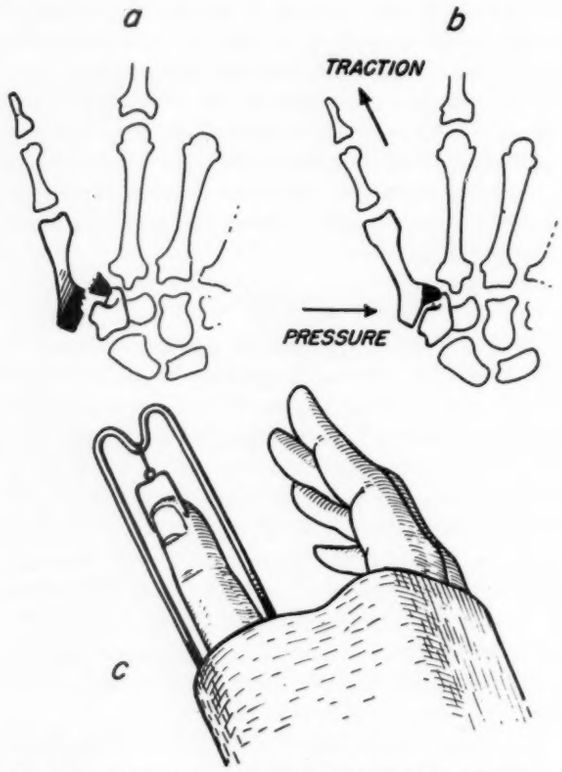


FIG. 21. Bennett's fracture of thumb. (*a*) Fracture dislocation of the carpometacarpal joint. (*b*) Reduction is by traction along the extended thumb with pressure over the base of the metacarpal. (*c*) Fixation is obtained by traction of the abducted thumb.

ment on the inner side. The metacarpal is displaced and slides down along the saddle of the greater multangular. This fracture should be reduced with traction and pressure over the base of the metacarpal. (Fig. 21.) A plaster gauntlet is applied, holding the metacarpal in extension and abduction, and traction is applied to the terminal phalanx. This fracture should be immobilized in traction for approximately four weeks, following which it should be immobilized without traction for one or two weeks until union is firm.

JOINTS

Strains of finger and thumb interphalangeal and metacarpophalangeal joints often have a

prolonged period of disability. These should be treated by immobilization for two or three weeks in a light plaster splint with the joint in a position of slight flexion. Following this period of immobilization active motion is instituted. Disability consisting of pain, swelling and limitation of motion may take months to disappear.

Dislocations of interphalangeal and metacarpophalangeal joints are usually due to hyperextension trauma with the phalanx being displaced backward. Reduction is usually accomplished by traction and the joint immobilized for two or three weeks in a plaster of paris cast in a position of function.

Dislocation of the metacarpophalangeal joint of the thumb is one of the most common dislocations of the hand. The phalanx is displaced backward, and the head of the metacarpal is driven forward through a split in the joint capsule which closes around the bone. The tendon of the flexor pollicis longus and the sesamoids, which are fairly constant on this joint, may aid further in locking the dislocation. This dislocation may be approached through an incision in the metacarpophalangeal crease of the thumb. Through this incision the volar plate of the joint capsule is reached and divided, separating the two sesamoids permitting the release of the head of the metacarpal.

Dislocation of the semilunar bone usually results from a fall on the dorsiflexed hand. The lunate bone is squeezed between the radius and capitate bones into the carpal tunnel, producing compression of the flexor tendons of the finger and the median nerve. Clinically there is limitation of motion of the fingers with median nerve palsy and swelling and thickening of the volar surface of the wrist. X-rays will reveal the diagnosis. If seen early they may be reduced by applying traction to the fingers and thumb, with pressure being applied to the volar surface of the dislocated bone. After manipulation the hand should be immobilized in about 45 degrees flexion for a week to ten days, following which a new cast should be applied with the wrist in neutral position for another two weeks. If the closed method fails, the lunate may be reduced through an incision made over the front of the wrist between the flexor carpi radialis on one side and the flexor tendons and median nerve on the other. Following this procedure the wrist is placed in flexion and incorporated in a plaster of paris cast. If old dislocations produce symptoms, complete excision of the lunate bone may be necessary.

There are many combinations of bones involved in the numerous dislocations of the wrist. In general, when these are fresh, they can be reduced by traction and manipulation. When these are old, open reduction is necessary to restore normal anatomy. Complications of these dislocations include degenerative arthritis and aseptic necrosis of the carpal bones, particularly the scaphoid.

SPECIAL INJURIES

Radiation Burns. Acute burns are usually the result of a single large dose. They are most commonly seen in hands of doctors or in patients after fluoroscopy. There is edema and erythema with deep boring pain. In severe cases the skin may turn white and slough; in milder cases the redness, swelling and pain disappear.[84]

In a case reported by Hempelman,[85] the hands became swollen within thirty minutes after massive exposure, corresponding to the equivalent of from 20,000 to 40,000 roentgen units of an 80 kilovolt x-ray machine. The right hand blistered thirty-six hours after exposure, and destruction of the skin continued in the right hand and forearm. During the second week, just prior to the patient's death, there was dry gangrene of the fingers of the right hand and of the left thumb.

Treatment of acute burns consists of sedation for the control of pain and some bland ointment such as cold cream or lanolin with a soft dressing.

Chronic radiodermatitis is manifested by atrophy, telangiectasis and keratosis of the skin. This may result in carcinoma. Treatment involves excision of the area with closure by a split-thickness skin graft or, if necessary, by flap grafts.

In reporting a series of such cases Daland[86] emphasizes the danger of giving any radiation to the hand of a growing child for fear of disturbing bone growth. He reported the case of a young girl who was treated with radium for hemangioma at the base of the right thumb. Radiation dermatitis developed at the site of radiation and the thumb grew to only half the length of its normal size.

Indelible Pencil Wounds. Dyes from indelible pencils may be analine dyes which are

FIG. 22. Hydrofluoric acid burn of right hand. Treatment with subcutaneous 10 per cent calcium gluconate.

especially toxic, causing necrosis of tissue. Such a wound heals very slowly. The initial treatment of these lesions is surgical. The whole of the discolored area should be excised and the resulting wound left open.

Grease Gun Injuries. Hand injuries produced by the injection of grease under pressure have been numerous. The mechanism is similar in all cases. A grease gun with a needle-like ejector is the usual offending agent. The operator accidently touches or comes very close to the end of the injector while the trigger of the gun is tripped. Grease is then injected into the finger or hand in varying quantities and under various pressures. It spreads diffusely throughout the tissue of the finger and hand, even going into the wrist and forearm. There is pain and swelling in the involved area due to the pressure of the mass, chemical irritation of the grease and secondary infection which may occur. Treatment at this stage involves use of penicillin to prevent secondary infection and sedation for the pain. Surgery should be limited to cautious removal of sloughing tissue or incision of fluctuant areas when abscesses develop. After the original wound has healed, the retained grease that is encapsulated in the chronic inflammatory tissue may cause the formation of oleomas. These are composed of fibroblasts, plasma cells, polymorphonuclear cells, lymphocytes, monocytes and giant cells. Treatment of these is surgical excision.[87]

Beryllium Granulomas. Beryllium produces various skin lesions such as dermatitis, cutaneous ulcers and subcutaneous granulomas. Flynn[88] has recently reported on two cases of subcutaneous beryllium granulomas of the hand in persons who had cut their hands on fluorescent light bulbs. The granulomas involved the tendon of the fingers which had to be excised with subsequent skin and tendon grafting. The granulomas consisted of fibroblastic tissue with lymphocytes and multinuclear giant cells. Although beryllium has not been used in the making of fluorescent light bulbs since 1949, Flynn emphasizes that there are still many of these in stock and caution should be exercised in disposing of the burned-out beryllium fluorescent lamps.

Hydrofluoric Acid Burns. Hydrofluoric acid, which has many uses in industry, causes a deep, slow-healing, painful burn. In severe cases there is noticeable destruction of tissue which may continue over a period of several days to weeks. Hydrofluoric acid has an extraordinary penetrative ability. It has been demonstrated that the intact hydrogen fluoride molecule is capable of penetrating intact epidermis. Figure 22 demonstrates a recent case treated at the Boston City Hospital. Such burns should be immediately washed with a warm solution of sodium bicarbonate and followed by a soaking of the part in alcohol. As soon thereafter as possible, under general or regional anesthesia, the soft tissues underneath the skin should be injected with 10 per cent calcium gluconate. This is done so that the fluorides in the tissues will be precipitated as insoluble calcium fluoride.[89] The previously mentioned case was treated in this manner and resulted in healing of the burned areas with epithelization. At present there is a thin, atrophic scar in the dorsum of the hand which may have to be excised and skin grafted.

Wringer Arm Injuries. This injury is incurred when an arm is caught between the rollers of a power-driven washing machine wringer. The involved extremity is contused or crushed, depending on the type of wringer. The involved area becomes swollen and ecchymotic; and if no treatment is undertaken, severe ischemic changes may occur in the hand and forearm. Devitalized skin will slough leaving granulating wounds. X-rays are usually taken but they rarely show fractures.

MacCollum et al.[90] have reported on 116 such injuries. They emphasize the importance of a sterile dressing together with a pressure dressing applied with an elastic bandage extending from the fingertips up to the axilla. The hand should

be placed in the position of function. Routine penicillin is recommended. The wound should be examined every day for the first few days to determine the extent of injury. If there are large accumulations of fluid under the tissues, they should be evacuated through a surgical incision. If the fascia covering the muscle bundles appears to be under severe tension, a fasciotomy should be performed. These wounds are sutured and a pressure dressing is re-applied to the entire extremity. When skin is lost, skin grafting should be done as early as possible. Active motion should be instituted early.

Goldner[91] has discussed severe "wringer" injuries which resulted in a clawhand deformity. If the deformity involves skin contracture only, it can be readily repaired with a skin graft. If the joint is functioning well but the extensor apparatus is destroyed, tendon grafting may be of value in restoring some active extension to the metacarpophalangeal joints. If joint function is limited, an attempt can be made through capsulotomy, dissection and stripping of the volar plate of the metacarpophalangeal joints to restore motion. When this is not possible, fusion of the metacarpophalangeal joints in the position of function will be necessary.

III. RECONSTRUCTIVE SURGERY OF THE HAND

Reconstructive surgery involving tendons and nerves has been discussed in the previous part devoted to trauma of the hand and will therefore not be discussed here.

Skin Contracture. Normal function of the fingers and hand may be prevented by contracture of the skin. Such contracture usually follows improper placing of skin incisions across flexion creases, keloid formation, severe infections, burns or tissue loss from trauma. The contracted tissue may involve skin, fascia, tendon, joints and intrinsic muscles.

The first stage of such repair requires removal of the underlying scar tissue with a plastic repair of the resultant defect. The replacement of skin will vary with the tissue defect. If this is limited, mobilization of the surrounding skin and subcutaneous tissue with primary closure may be satisfactory. Sliding flaps from adjacent areas may close the defect. This is of particular value if the defect is on the palmar surface of the finger and hand, and the tissue can be obtained from the lateral areas. In this case the donor site will be closed with a split-thickness skin graft.

Flexion contractures of finger webs and fingers can often be corrected by Z-plasty procedures. When the surface replacement does not involve deep tissues and the defect is large, free skin grafts are useful. As a general rule, split-thickness skin grafts are satisfactory for the dorsal and lateral areas of the palm and fingers. Full-thickness skin grafts or deep split-thickness skin grafts are used on the palmar surfaces. It is essential that the edges of the graft do not cross flexion creases but are placed lateral to such creases. When the defect obtained by removing the contractured skin and scar tissue is deep, involving tendons and nerves, it must be closed with flaps. Pedicle flaps are useful but there is an increasing tendency to use single flap grafts. It is essential that these grafts be kept closed to avoid an open wound with subsequent edema and increased fibrosis.

Contracture of the web space between the thumb and index finger may be considered as a special topic. Several excellent articles have been written concerning this subject.[92–94] Maximum spread of the thumb is obtained when the thumb metacarpal is 45 degrees forward from the transverse plane of the palm. Wounds or incisions along the web of the thumb (Fig. 23A) or between the base of the thumb and the thenar crease (Fig. 23B) will result in contractures. After excision of the contractured area the skin can be replaced by either a Z-plasty or local shifting if the defect is small. In most cases in which the palmar surface of the hand is involved a pedicle or direct flap will be necessary, taking care that the web space is covered from hinge to hinge. If the scarred area involves only the dorsum or is not particularly deep, it may be replaced by a split-thickness skin graft, taking the same precautions in placing it.

Figures 24A and B show the right hand of a truck driver who suffered a severe burn of the palm and volar surface of the fingers and thumb. The initial treatment was not satisfactory in that the web space of the thumb and index finger was not maintained, and skin grafting was performed too late. As a consequence, flexion contracture of the index, middle

and ring fingers and an adduction contracture between the thumb and index finger developed. A tube pedicle graft was raised from the left thoracicoabdominal region. When it was thought that this was sufficiently vascularized to shift, the scar tissue was excised from the

FIG. 23. Contractions in the line (A) between thumb and hand will result in adduction deformity of thumb, and along line (B) will result in limitation of extension of thumb.

palm of the hand and web space of the thumb and index finger, and the flap of skin inserted as shown in Figure 24C. At a suitable interval following this the tube graft was removed from the abdomen and tailored to fit the wound. The result is shown in Figure 24D and E. A suitable web space was developed and the patient can now resume his former occupation. There is still limitation of motion in the fingers due to the prolonged immobility in the previously shown position.

Dupuytren's Contracture. This is a hyperplasia of the palmar fascia which occurs in about 2 per cent of the population. It is commonly bilateral and may be associated with a similar involvement of the plantar fascia and rarely with Peyronie's disease. It may occur at any age but is most common between thirty and seventy. It occurs about nine times as frequently in males as in females.

The etiology is disputed. Most authors agree that heredity has definite significance. Repeated minor trauma cannot be ruled out as a cause, however, since it is present in a large percentage of the histories obtained from patients with this disease. Skoog[95] found that the incidence of contracture was 42 per cent in a group of male epileptics. Steinberg[96] believes that it is a type of fibrositis. Clay[97] believes that in a certain percentage of cases it is a true neoplasm, a cellular fibroma of the palmar fascia.

The occasional occurrence of this contracture as a sequel to coronary artery disease must be remembered. Askey[98] reported seven of ten cases of postinfarctional shoulder and hand syndrome with contracture of the palmar fascia similar to Dupuytren's. Kehl[99] reported six cases following coronary occlusion. In three cases the changes appeared bilaterally. Although the pathogenesis of this relationship is not clear, a review of the cardiac status of every middle-aged and elderly patient who is to undergo surgery for Dupuytren's contracture should be undertaken.

Regardless of the cause, the palmar fascia undergoes continuous thickening involving the overlying volar skin and all the prolongations of the palmar fascia into the fingers, with consequent flexion contractures of the proximal and middle joints of the involved fingers. Microscopic examination shows fibrous hyperplasia of the palmar fascia.

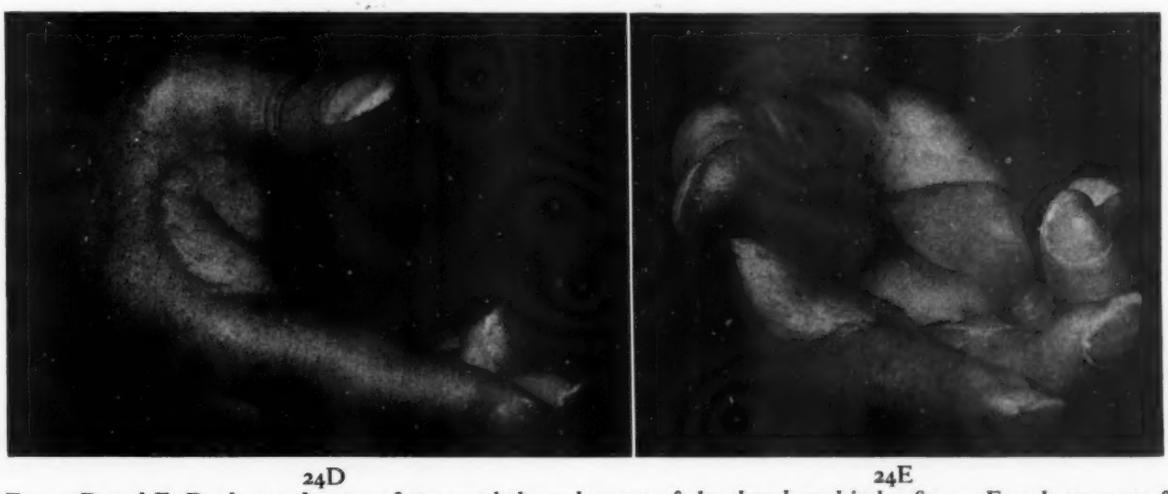
Clinical development takes place at varying speeds. Some cases never progress over thirty or forty years, and in others there is severe flexion which develops within several years. The disease usually begins as a thickening or nodule in the skin of the palm just above the base of the ring finger associated with puckering of the skin. As it progresses it causes a flexion contracture of the ring finger. (Fig. 25.) Less commonly involved are the fifth finger, middle finger and thumb. In Ross and Annan's series[100] the fingers were involved as follows: ring finger, forty-five; little finger, forty; middle, twelve; thumb, three; index, none. Dorsal knuckle pads or nodules over the proximal interphalangeal joints of one or more of the fingers are seen which are similar histologically to the palmar contracture.

Treatment of this condition requires complete surgical excision of the palmar fascia. The operation is always performed in a bloodless field, and the type of incision varies with

24A 24B

FIG. 24A and B. A, adduction contracture of volar aspect of thumb due to burns. B, dorsal aspect demonstrating adduction contracture.

FIG. 24C. Tube pedicle graft raised from abdomen and applied to palm of hand.



24D 24E

FIG. 24D and E. D, the resultant graft restored the web space of the thumb and index finger. E, volar aspect of restored hand.

FIG. 25. Dupuytren's contracture, right palm, demonstrating flexion deformity of fourth and fifth fingers.

the author. The Hand Service at the Boston City Hospital has recently been using two incisions; one extending along the distal palmar crease and the other following the curve of the thenar eminence. During the dissection of the fascia great care must be taken that all the extensions of the fascia between the tendons and neurovascular bundles be removed. When the proximal phalanx of the finger must be approached, it may be done through either a flap or a Z-plasty incision. If the vascularity of the skin flaps is compromised, suitable excision of skin must be performed with grafting of a full-thickness skin graft into the palm. Following this the hand is placed in the position of function and a pressure dressing applied for a week or ten days, following which active motion is instituted. In an attempt to avoid postoperative hematoma with consequent stiffening of the hand, Tanzer[101] has advised the use of a compression suture as follows: "Before the skin incisions are closed, a mattress suture of #000 silk, double-armed on straight needles and incorporating a button, is passed directly through the hand at two points. The first needle penetrates the skin between the second and third metacarpal bones, passes under direct vision to one side of the neuro-vascular bundle and pierces the intermetacarpal space at a level far enough distalward to avoid the deep volar arch and ulnar nerve emerging through the dorsal skin. The second needle is introduced in the same manner between the fourth and fifth metacarpal bones. After the hand closure is complete, packing is carefully introduced beneath and around the silk loop in the palm. The button is drawn into the packing by gathering up on the dorsum of the hand the two suture ends which are threaded through a second button and tied with moderate firmness over gauze to maintain snug pressure."

In the elderly patient it may not be advisable to perform the radical operation for fear of limitation of motion of the hand, and an excision limited only to the involved fascia may be of value.[102] Subcutaneous fasciotomy may offer palliative relief to such persons.

Steinberg has claimed relief from this disease by treatment with vitamin E; however, King[103] could find no evidence of benefit by this treatment.

Ischemic Contracture. Ischemia of the forearm or hand, for a temporary period of time, may result in destruction of muscles with subsequent fibrosis and contracture. When the muscles of the forearm are involved, such a state is known as Volkmann's contracture. This most commonly follows a supracondylar fracture of the humerus or fracture of the bones of the upper forearm but may follow any trauma to the arm. It has been seen following embolism. Although this entity was first believed to be caused by venous obstruction in the antecubital fossa, it is now believed that the prime factor is arterial insufficiency.[104,105]

The clinical picture begins with pain in the forearm and hand associated with edema and cyanosis. Hypesthesias and paresthesias may

appear in the distribution of the nerves in the hand. The radial pulse is usually absent. Exploration of the forearm at such a stage discloses swollen, necrotic muscles with profound arterial damage or spasm. If the disease progresses, these degenerated muscles become atrophic and contracted, and the fingers and wrist assume a flexed position. Attempts to extend the fingers result in severe pain in the forearm. As the disease process continues, there will be limitation of motion in the joints in the hand due to disuse. Nerve lesions may be severe with the usual manifestation of nerve injury.

Treatment of this condition may be divided into the immediate and late treatment. As soon as this condition is diagnosed, every effort should be made to restore the circulation in the involved extremity. If the elbow fracture was placed in acute flexion, the elbow should be extended to see if the radial pulse would be restored. Following this the antecubital space should be explored to examine the arterial system. If the artery is completely destroyed, arteriectomy should be performed. Sympathectomy may be of value in improving the circulation, as has been observed by Foisie.[106] Crystal et al.[107] have recently presented a case in which a thrombectomy was performed twenty hours following injury. Following this procedure on the brachial artery, circulation was restored in the distal arterial supply. There was almost a complete cure of this condition.

Cregan[108] presents a case of prolonged arterial spasm of the brachial and ulnar arteries which was explored nine hours following a supracondylar fracture of the humerus. Examination showed the arteries to be in tight spasm with no localized arterial injury. All the smaller arteries were collapsed, but the veins and nerves appeared normal. The main arteries were mobilized and swabbed with novocain. The color of the hand improved appreciably but the arteries showed no more than slight relaxation. No pulsations were seen. The wound was closed, the brachial plexus infiltrated with novocain and the fracture reduced. The hand gradually became pink and warm but the radial pulse did not return for twelve hours. Six months following this there was no evidence of ischemic muscle contracture or nerve lesions.

There is another school of thought on the treatment of the acute condition. Seddon[109] has had experience with immediate operation on the forearm and excision of necrotic muscle. In this way it is believed that the contracture of the necrotic muscle would be prevented and the disability thereby decreased. The difficulty with this, however, is that one is not sure of the muscle regeneration. For instance, Horn and Sevitt[110] followed the regeneration of a tibialis anterior muscle by muscle biopsy for weeks following a rupture of the popliteal artery. At the beginning the muscle is brown, dry, friable and quite avascular. Histologic section of this muscle nineteen weeks after injury showed necrotic muscle with absence of nuclei and discoidal fragmentation. A second biopsy fifty-seven weeks after injury showed no necrotic muscle in the section. Essentially a regeneration of muscle took place, pointing out the regenerative power of human muscle and presenting the problem of deciding what muscle to excise at the time of initial surgery. Griffiths[109] suggests that perhaps the response to intravenous curare or the persistence in the muscle of injected radioactive substances might be a useful guide. Further experiments should be carried on along this line of reasoning.

For the late condition, a long period of physiotherapy with dynamic splinting to overcome the contractures should be followed. Efforts should be made to restore motion in all the joints of the fingers, hand and wrist. If this is not sufficient to overcome the deformity, some operative procedure may be necessary. Tendon lengthening of the contracted tendons may be done. Nisbet[111] has described a muscle-stripping procedure in which the contracted muscles are stripped from their origin in the forearm permitting the shortening to be overcome.

Parkes[112] describes an operation in which the flexor carpi radialis, palmaris longus and flexor carpi ulnaris are completely divided at the wrist to permit dorsiflexion. Tendons of the flexor digitorum sublimis are resected, and the tendons of the flexor digitorum profundus are divided high enough above the wrist to avoid retraction of the distal stumps into the carpal tunnel when the fingers are fully extended. The tendon of the flexor pollicis longus is similarly divided to permit extension of the thumb. The tendon of the extensor carpi radialis longus is divided at its insertion and freed for a considerable distance from the extensor carpi radialis brevis. This muscle with its tendon is re-routed in as straight a line as possible from

its origin to the front of the wrist, where it is buttonholed through the four tendons of the flexor digitorum profundus and fixed in the usual way. Correct tension is made at the suture line to insure that full advantage is taken of wrist movement. This operation is really a

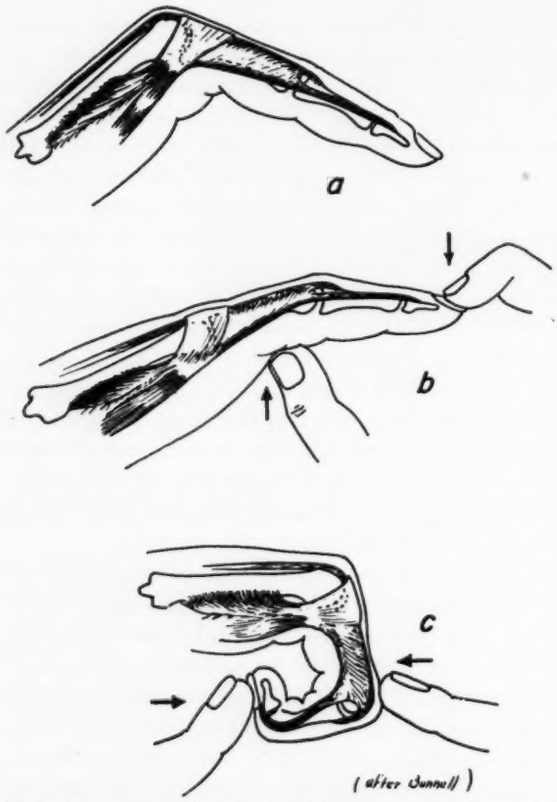


FIG. 26. "Intrinsic plus" deformity of finger. (*a*) Characteristic position of finger; (*b*) forced extension of metacarpophalangeal joint prohibits flexion of finger; (*c*) flexion of metacarpophalangeal joint permits flexion of finger.

tenodesis and it restores flexion of the fingers by means of the trick movement of dorsiflexion. It is interesting that the lack of voluntary power of the thumb produced by this operation causes no material disability providing there is active opposition of the thumb.

It is of interest that Thompson and Mahoney[113] have described a contracture similar to Volkmann's contracture in the lower extremity following fracture of the femur.

Bunnell[68] has described a similar condition occurring in the intrinsic muscles of the hand following encasement of hands in plaster of paris or tight elastic bandages. He describes it as *ischemic contracture, local, in the hand.* This contracture may follow damage to the great vessels and may accompany Volkmann's ischemic contracture or may occur entirely independent of it. There is a characteristic posture of the hand with the metacarpophalangeal joints held in flexion and the interphalangeal joints in extension with the straight thumb in the palm. The characteristic deformity is due to contraction of the interosseus and lumbrical muscles. This position has been described by Bunnell as the "intrinsic plus" position. If the metacarpophalangeal joints are passively forced into extension, the distal interphalangeal joints cannot be flexed. (Fig. 26.) However if the metacarpal joints can be held at flexion, these distal joints can be flexed. In mild cases a splinting of the hand may overcome the deformity. However, in most cases operation will be necessary. If some interosseus function is still present, Bunnell suggests that the interosseus muscles be separated from the metacarpals by periosteal dissection and allowed to advance distally to permit extension of the metacarpophalangeal joints and flexion of the two distal joints. On the other hand, if the intrinsic muscles are too fibrotic to function or if previous stripping has been unsuccessful, tenotomy of the lateral bands of the fingers should be performed through a short lateral incision on the dorsal aspect of each of the three finger webs until the fingers can be fully flexed with the metacarpophalangeal joints extended.

When such a condition involves the web space of the thumb, there will be an adduction contracture. Various procedures may be necessary to restore normal function. The contracted skin should be excised and the intrinsic muscles stripped from the thumb. After the defect is skin grafted from hinge to hinge, the full thumb spread may be maintained by transfixing the metacarpals of the thumb and index finger with two Kirschner wires. At a later date, tendon transfer to restore opposition to the thumb may be indicated.

Joint Contracture. Immobilization of the hand for a prolonged period of time results in stiffening of the finger joints. This may be seen in any of the aforementioned injuries. When the metacarpophalangeal joints are affected, some form of movement in the joints may be restored by either a capsulotomy or arthroplasty. Capsulotomy should be successful if there is no joint disease. X-ray should be taken of all such joints before surgery is attempted. Capsulotomy consists of excision of the collateral

ligaments of the joint. If passive motion of the joint is less than expected, the anterior capsule should be explored; and if it is adherent to the bones, it should be freed by blunt dissection. A transverse incision of the capsule should not be done because subluxation may occur, and there may be a greater degree of ankylosis than before operation. If the joints are diseased, some form of arthroplasty is indicated providing the skin and soft tissues are in good condition. Either the metacarpal head or the proximal centimeter of the proximal phalanx is resected, and a strip of fascia is sutured over the proximal bone so that its gliding surface faces the flexor and extensor tendons around the joint.[114]

When the proximal and distal interphalangeal joints are ankylosed, capsulotomy is rarely successful. There is probably no place for arthroplasty because of lateral instability in these joints. The most useful procedure is an arthrodesis at an angle of moderate flexion. Occasionally amputation of a hopelessly ankylosed finger may be necessary.

If the interphalangeal and metacarpophalangeal joints of the thumb are involved, an arthrodesis in the position of function is often the best procedure. When the carpometacarpal joint of the thumb is involved, several procedures may be of value. A capsulotomy of the carpometacarpal joint, in addition to other procedures on the soft tissues, may restore function as suggested by Howard.[94] If the navicular bone moves normally, an arthrodesis of the carpometacarpal joint may give a useful digit. However, if the navicular bone is also ankylosed on the carpus, an arthroplasty of the carpometacarpal joint may be of value.

The carpometacarpal joint of the thumb is a common site of osteoarthritis in the hand. In the presence of this disease x-rays will show narrowing of the joint space, subchondral condensation of bone, lateral subluxation, osteophyte formation and deformation of the medial and distal angle of the multangular. If the symptoms are sufficiently severe, Müller[115] suggests that arthrodesis of this joint be performed. Gervis[116] presents the opposite point of view and believes that excision of the greater multangular is the procedure of choice for this disease. He reported fifteen cases in which this operation was performed. The result was full and painless movement of the first metacarpal bone.

Bone Deformity. Fractures of the fingers may heal with malunion resulting in poor function of the fingers and hand. Treatment will consist of either osteotomy and correction of the deformity or amputation of the finger. Of particular importance are the fractures of the thumb and index finger which may result in a narrowing of the thumb web space. Malunion of Bennett's fracture may result in such a web space narrowing. Treatment consists of bone reconstruction doing either an osteotomy or a fusion of the carpometacarpal joint. Fractures of the proximal half of the index metacarpal with malunion may give a relative contracture of the thumb web. Treatment may consist of osteotomy with proper bone alignment.

Reconstruction of Digits. Destruction of the thumb is one of the most disabling of all hand injuries. Considerable attention has been devoted to reconstruction of this digit. If there is adequate length of the thumb stump and normal flexion of all the fingers, the quickest and simplest procedure is a deepening of the web space between the thumb metacarpal and index metacarpal such as one would do for syndactylism.

It may be useful, however, to restore length to the thumb. Lewin[117] uses an iliac bone graft inserted into the stump of the metacarpal to restore length. Normal sensation in the digit is obtained by elevating the original skin around the thumb. The resultant defect over the thenar eminence is covered by a split-thickness skin graft. Thumb length may also be restored, as suggested by Greely,[118] by inserting a piece of rib into the metacarpal of the thumb following a tube pedicle graft of skin and subcutaneous tissue to the thumb stump. This procedure has a disadvantage since normal sensation is not present in the thumb. Pollicization of an adjacent finger has been performed by several surgeons.[119,120] Marble[121] believes that such pollicization should be done only with a relatively deformed finger and not with a normal finger. Clarkson[122] has had experience with numerous cases in which he has transferred the great toe to the hand to replace a thumb.

Loss of all five digits of the hand is an extremely crippling deformity. Graham et al.[123] have described four cases of such deformity wherein they created a "lobster claw" type of hand by elongating the first metacarpal, if

necessary, excising the shaft of the second metacarpal leaving only the base, creating a cleft between the third and first metacarpals by bringing a rectangular flap of skin from the dorsum of the hand through the cleft and suturing it to the base of the palmar wedge. The palmar skin obtained by excising the second metacarpal is sutured dorsally into the cleft onto the opposing surfaces of the first metacarpal. Higgins[124] has described a procedure wherein the second and third metacarpals, the lesser multangular and the body of the capitate bone are excised for a similar condition. Since an S-shaped incision was used for the procedure, the first metacarpal is covered with palmar skin and the third and fourth metacarpals with dorsal skin. The resultant stump in this individual had surprisingly good grasping function and was quite suitable for manual labor.

IV. CONGENITAL DEFORMITIES

Congenital deformities consist of absence, fusion, multiplicity and altered growth of parts of the upper extremity. They have been well discussed by Bunnell[23] and Barsky.[125] A few of the deformities will be presented in this review.

ABSENCE OF PARTS

Paraxial Hemimelia. Congenital absence of the radial or ulnar side of the hand may take place. In the defect of the radial bud there will be absence of all or part of the radius, the radial bones of the carpus (scaphoid, trapezium, first metacarpal) and often the thumb and adjoining finger together with defects in the soft parts along the radial side of the forearm. The ulna is short and thick; and as the hand grows, it deviates toward the radial side of the palm until it is often at a right angle to the forearm. Treatment involves proper splinting during growth to correct the deformity with subsequent excision of contracted parts and fusion of the hand in correct alignment.

A defect of the ulna bud, which is less frequent than the radial, may give absence of part or all of the ulna, together with the pisiform, hamate and some of the other metacarpals and associated digits. Defects in the soft tissues on the ulnar side of the arm will take place. Since the radius is the most important bone in the wrist, the hand dysfunction is not as severe as with the opposite defect. Treatment is instituted according to the principles outlined for radial hemimelia.

As previously mentioned, paraxial hemimelia may occur only in the soft tissues. White and Jensen[126] have recently reported a series of cases in which the extensor pollicis brevis is absent bilaterally, with the resultant deformity of the first metacarpal bone held in abduction and the proximal phalanx flexed forward and partially subluxed. There is also ulnar deviation of the hand due to absence of this tendon. These cases are repaired by transferring the common extensor tendon of the index finger to the dorso lateral surface of the proximal phalanx of the thumb.

Cleft Hand. This is a rare anomaly in which there is a defect of the central portion of the hand. A V-shaped cleft, which tapers proximally, dividing the hand into two parts is the usual finding, the digits in each part being webbed. The middle metacarpal is usually missing. Treatment of this condition may benefit the patient. If the cleft is too wide, its function may be improved by narrowing it. If the person's hand is functioning with a "pincer-like" movement, it is probably more important to keep this movement rather than try to improve the appearance.

Annular Groove. Congenital annular grooves may be present anywhere in the upper extremity. They may be superficial involving only skin and subcutaneous tissue, or they may involve fascia, tendons and bones, resulting in amputation of the part. They are usually combined with some other anomaly such as syndactylism and brachydactylism. Treatment, when indicated, is directed at excising the circular bands and reapproximating subcutaneous tissue and skin. To prevent recurrence of the constriction, the skin incision should be made so that a circular scar does not occur. This may be done by making alternate diagonal cuts on each skin margin down to the deep fascia, thus doing a modification of a Z-plasty. One probably should not encircle the finger completely at one operation but should do only half a finger at a time.

FUSION OF PARTS

Syndactylism. In this disease there is a joining of one or more of the fingers, the middle and ring fingers being most commonly in-

FIG. 27. Syndactylism.

volved. (Fig. 27.) All the fingers may be webbed, giving a "mitten-like" appearance to the hand. This affliction is twice as frequent in males as in females and is often associated with other defects such as polydactylism. Operation is usually performed when the child is three or four years of age unless it is believed that the webbing is altering the growth of the bones. The classic operation is the one first suggested by Zeller in 1810. In this the fingers are cut apart and a V-shaped flap is made on the web space of the dorsal and palmar surfaces. The two flaps are sutured side by side, thus developing an adequate web space. The sides of the fingers are covered by a skin graft, in which case the skin graft covering the web should extend onto the dorsal and palmar surfaces. If a double nail is present, it should be split and a portion of the nail and its matrix on each side removed, so that a skin graft may be brought over and around the nail margin. If more than two fingers are fused, Barsky suggests that one has to be cautious about separating both sides of a digit. It is probably safer to operate on one side of the finger at a time. If the tendons are common, they should be left attached to the better finger with a tendon graft being performed on the other finger.

Sympbalangism. This is a fusion of the interphalangeal joints, usually the middle one. An arthrodesis of the joint in the position of function is the procedure of choice if an operation is deemed advisable.

Synostosis. Fusion of other bones of the arm may occur. Probably the most numerous are those in the carpal region. O'Rahilly[127] has made a survey of carpal anomalies. Practically every conceivable combination of carpal fusion has been reported. The most common is the lunate-triquetral fusion. For some reason this appears to be more common in the African race. Minnaar[128] reported a series of twelve such fusions which he had personally seen in members of the South African Bantu.

MULTIPLICITY OF PARTS

Hyperpbalangism. This deformity consists of an excessive number of phalanges, the thumb being most commonly involved with three phalanges present.

Accessory Bones. O'Rahilly[127] has studied and reported twenty-five such bones and has attempted to standardize their terminology.

ALTERED GROWTH

Congenital flexion contracture of fingers most often involves the fifth finger. The proximal interphalangeal joint is usually held in flexion with the other two finger joints held in extension. Bunnell[23] states that the surgical treatment of this is very disappointing, since all the tissues are involved in the contracture and it tends to recur after their excision.

Brachydactylism is a condition in which some of the phalanges or metacarpals of digits are short or absent. It is often associated with other congenital defects of the hand. Shoul and Ritvo[129] report an interesting series of three cases of brachydactylism in which the involved fingers had four phalanges.

Madelung's deformity occurs when the growth of the radius falls behind that of the ulna, pro-

ducing a volar and ulnar curve of the radius. The increased growth of the ulna makes this bone luxate from the radius and project dorsally and distally, giving a clinical impression of a forward dislocation of the wrist. There is also limitation of motion in the wrist. Treatment is deferred until bone growth is completed. Following correction of soft tissue contracture the head of the ulna is removed and a sling placed around it to prevent its dislocation. The forearm is then splinted in an overcorrected position. If the radial deviation is sufficiently severe, a wedge osteotomy may be necessary.

Megalodactylism consisting of hypertrophy of the digits seldom is benefited by surgery.

V. TUMORS OF THE HAND

Practically every known tumor can occur in the hand; therefore it will be the purpose of this review to comment on the tumors more commonly found in this region.

Ganglia are cystic tumors which occur about the joints of extremities. They are probably a mucoid degeneration of the connective tissue of the joint similar to adventitious bursas.[130] Trauma may be a factor in this degeneration. They are composed of a fibrous, connective tissue wall with contents resembling soft jelly. They never become malignant. They are most commonly observed about the wrist joint on the dorsal aspect between the long extensor of the thumb and index finger. The next most common site is on the volar aspect of the wrist between the tendons of the flexor carpi radialis and the brachioradialis. In this particular location care must be taken to avoid damaging the radial artery at time of surgery. They can also arise on the deep pulleys over the long flexor tendons of the fingers and on the dorsum of the interphalangeal joints. Treatment is indicated when they cause pain or cosmetic deformity. Surgical excision should be done, taking care to remove a flange of joint capsule around the base of the tumor.

Epidermoid cysts or implantation cysts occur in people who traumatize their hands. They are commonly seen in laborers, carpenters, gardeners, etc., and are present on the palmar aspect of the hand or fingers, confining themselves solely to the skin. It is believed they are due to implantation of surface epithelium into the deeper layers of the skin. On microscopic section the walls of these cysts are composed of fibrous tissue with an inner layer of squamous epithelium which may show laminated keratin. Treatment is complete surgical excision.

Sebaceous cysts may occasionally be present on the dorsum of the hand or fingers. They are never seen on the palmar surfaces since sebaceous glands do not occur in this area.

Mucous cysts of the skin are myxomatous lesions believed to be due to mucoid degeneration of the skin. They are usually present on the dorsal aspect of the distal interphalangeal joint of the fingers, the long finger being most commonly involved. (Fig. 28.) The great majority of these occur in women. Since they arise in the skin, the skin cannot be moved over them. They appear as translucent painless nodules and may be multiple. Microscopically, the wall is made up of collagen fibers which send thin, irregular trabeculae toward the center of the cyst producing a lace-like pattern in which mucoid material is seen. The recurrence rate following surgery is high unless a margin of skin is removed around the lesion. The resultant defect will have to be skin grafted.[131]

Glomus tumors are usually small, purplish blue lesions characterized by excruciating pain. The slightest pressure on this lesion may cause severe pain. They arise from the glomus, a normal end organ consisting of an arteriovenous anastomosis containing smooth muscle cells and large clear cells referred to as epithelioid or glomus cells. The glomus controls arteriovenous circulation within the fingers and extremities and controls the temperature of the body. These organs are distributed widely over the body. They occur most frequently in the nail beds and fingertips and secondly in the volar aspects of the first, second and third phalanges of the upper and lower extremities. The tumors are usually very small, being a few millimeters in diameter, and if not visible may be picked up by the pin test.[132] However, they have been reported up to 3 or 4 cm. in diameter.[133,134] The subungual glomus, which is the most common variety, may occasionally erode the bone of the underlying phalanx.[135,136] Treatment is surgical excision, and recurrences following surgery have not been reported.

Giant cell tumors of tendon sheaths (xanthoma) occur most often in the digits as clearly defined

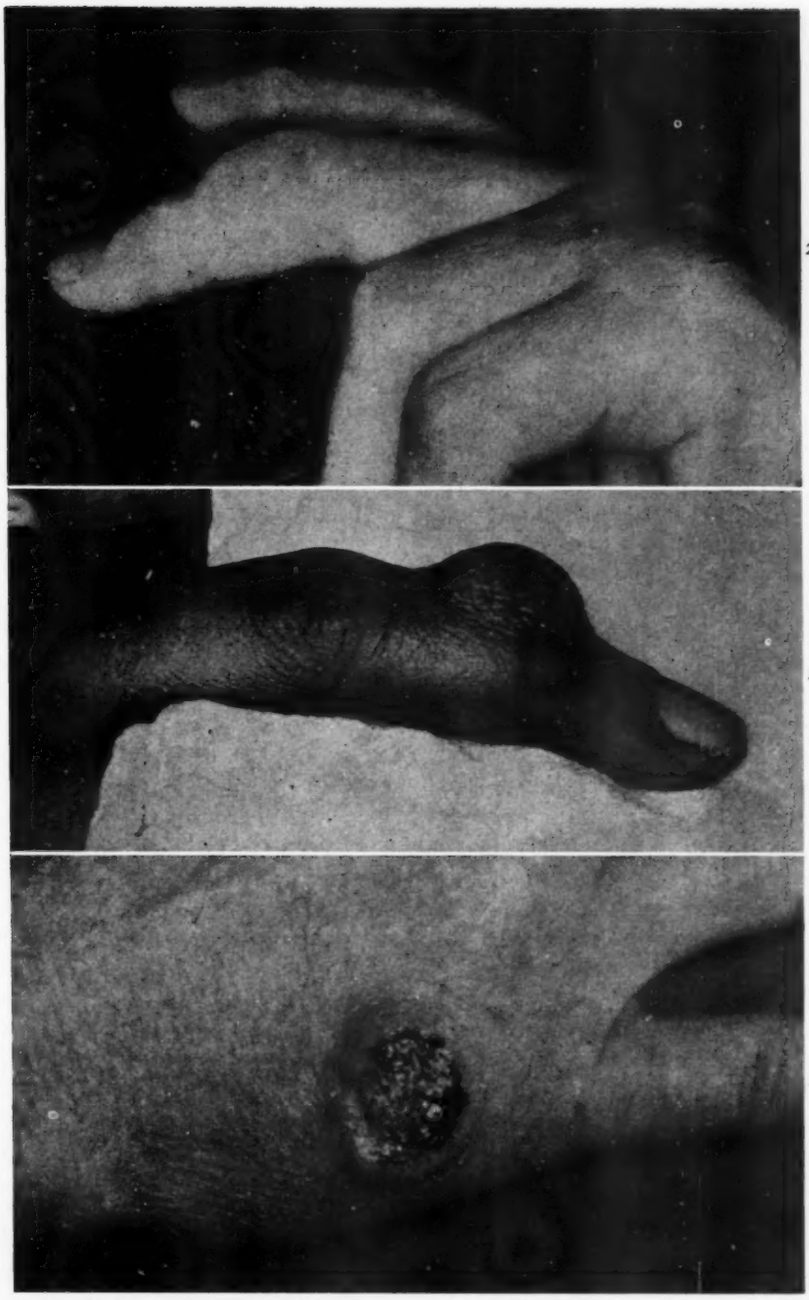


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FIG. 28. Mucous cyst, left third finger. Fig. 29. Giant cell tumor. FIG. 30. Epidermoid carcinoma, dorsum right hand.

round nodules 1 cm. in diameter. (Fig. 29.) They involve the volar and dorsal aspects of the fingers with equal frequency. There is a peak incidence in the fourth decade, women being afflicted more than men. Microscopically, cholestrol deposits may be present in a stroma of deeply eosinophilic connective tissue containing giant cells and occasional foam cells. They may occasionally erode bones. Treatment is surgical excision.[137]

Carcinoma may occur in the hand and upper extremity, (Fig. 30.) Clarke[138] has reviewed 214 cases of primary carcinoma of the extremity seen at the Barnard Free Skin and Cancer Hospital from 1925 to 1940. This comprised 4 per cent of all the cases of skin cancer seen

during this period. Eighty-five per cent of these cases occurred in the upper extremity, the majority occurring in males. The maximum incidence took place in a group of patients seventy years of age or older. The great preponderance of lesions presented themselves on the dorsum of the hand. There appeared to be a definite relationship to exposure to the sun, since 163 of the lesions of the upper extremity were squamous cell carcinomas and eighteen were basal cell lesions. Treatment was by wide local excision or amputation. The decision to amputate depended on the fixation of the lesion. If lymph nodes were felt in the epitrochlea region or in the axilla, an axillary node dissection was performed. The prognosis in this series was good with regard to the upper extremity. In a follow-up of 136 cases there was a survival rate of 93 per cent when the lymph nodes were not involved. In eleven axillary dissections revealing positive nodes the survival rate was 66 per cent.

Cartilaginous tumors of the hand have been recently reviewed by Shellito and Dockerty.[139] Forty-two cases found at the Mayo Clinic were reviewed, thirty being enchondromas and twelve ecchondromas. The enchondromas were found in the small bones of the phalanges in twenty-five cases and in the metacarpal bones in fifteen cases. Two were found as tumors of tendon sheaths. Five of the tumors were multiple. X-rays of the enchondromas revealed the so-called bone cyst with the cortex expanded and thinned.

The ecchondromas occurred with equal frequency on the phalanges and metacarpals at the insertion of a tendon usually on the proximal side. X-rays showed the elevation of bony structure appearing at one end of a bone.

Treatment of both enchondromas and ecchondromas is conservative and limited to surgical excision. If there is not sufficient bony shell left in the shaft of a bone, bone grafting may be necessary.

Osteoid osteomas are bone lesions characterized by pain and a definite x-ray appearance consisting of radiolucent central nidus with a surrounding osteosclerosis. The central nidus may be a few millimeters in diameter. Since Jaffe[140] described these lesions in 1935, much controversy has arisen concerning their true nature. They have many of the elements of inflammation and neoplasm. Carroll[141] has recently reported six cases of osteoid osteoma found in the hand and collected twenty-two cases from the literature. These lesions were all cured by surgical excision.

Pyogenic granulomas consisting of friable granulation tissue and often resembling tumors may occur at the site of wounds and incisions. Treatment is by excision or cautery.

VI. VASCULAR DISEASES OF THE HAND (TRAUMA)

Trauma may disturb the circulation of the hand by direct injury to an arterial trunk or by reflex sympathetic vasospasm. Vascular injuries in the upper extremity comprised about one-third of all the vascular injuries in World War II and the Korean War. In World War II gangrene requiring amputation of an extremity occurred in 26.5 per cent of brachial artery injuries, in 43 per cent of axillary artery injuries and in 28.6 per cent of subclavian artery injuries. The results following brachial artery injuries depend on whether or not the profunda branch is intact. Injuries above the profunda resulted in a 55 per cent amputation rate, and below the profunda in a 25.8 per cent amputation rate.[142]

Jahnke and Seeley[143] reported on a series of seventy-seven injuries that occurred in the Korean War. They approached the vascular injury through classical surgical incisions described by previous writers and, if possible, did not go through the initial wound. About 1 cm. of lacerated artery was removed from each end, since examination of such tissue demonstrated histologic changes due to trauma even at this distance from the original injury. The artery was well mobilized and the adventitia was stripped from the artery proximally and distally for many inches, in some cases up to 10 inches. When necessary, important collateral branches were divided to permit mobilization. Partial flexion of extremities was utilized to permit sutures without tension. The usual Carrel everting anastomosis was used. It was deemed imperative that the anastomosis be covered with muscles or fascia at the completion of the procedure! Using this technic, a vein graft was needed in only four of the reported cases. The result of this method of treating the traumatic vascular injuries was

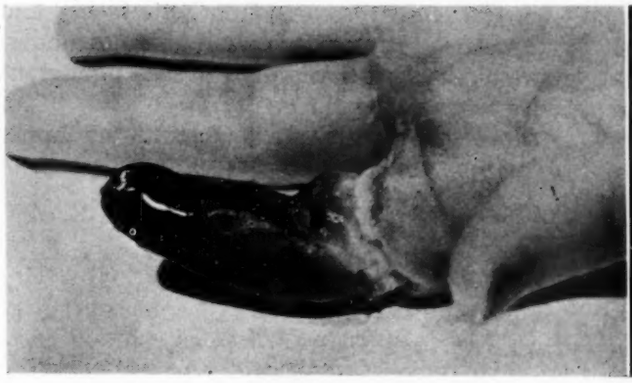

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FIG. 31. Digital gangrene resulting from digital nerve block with 1 per cent novocain solution with epinephrine.

FIG. 32. Gangrene resulting from levophed infusion in forearm.

extremely gratifying, since no gangrene occurred in the major arterial injuries of the upper extremity.

CHEMICAL GANGRENE

Ergot preparations have long been known to produce gangrene in extremities. Cases of digital gangrene of the upper extremity have been reported following the use of ergotamine tartrate.[144] Contraindications to the use of this drug are pre-existing sepsis, obliterative vascular disease and cardiovascular disease. It should be employed with caution in cases of hepatic or renal disease and hypersensitivity to the drug.

Digital nerve block occasionally results in gangrene of a finger. This is usually due to epinephrine in the novocain solution, but secondary factors such as the use of a tourniquet or too much injected solution may play a role. When performing a digital nerve block for anesthesia of a finger, no more than 1 to 1½ cc. of 1 per cent novocain should be used for the block with no tourniquet being used.[145] Figure 31 shows a previously unreported case of digital gangrene following a digital block in which epinephrine was used in the novocain solution. This brings the total number of such cases personally seen by the author to nine, eight of which have been previously reported.[145]

Phenol dressings were frequently used on fingers and toes in the past but resulted in so many cases of gangrene that their use has been discontinued.

The present use of levophed® solution will occasionally result in gangrene at the site of intravenous infusion. Figure 32 demonstrates a patch of gangrene resulting from a levophed infusion. A plastic catheter was placed in a vein and introduced about 10 inches proximally in the arm. At the site at the tip of the catheter an area of gangrene was produced following infusion of this solution.

FROSTBITE

Frostbite will result in amputation of digits in approximately 10 per cent of military cases.[146] It may result from one or more of several factors: direct injury from freezing, edema of the hyperemic phase occurring soon after thawing and vascular occlusion by agglutinative thrombi.

Much experimental work has been done in searching for a better method of treatment of this condition. Heparin was suggested to prevent the vascular changes but results are conflicting. Recently, Lewis and Moen[147] reported that heparinized experimental animals showed as much gangrene as other animals, and the death rate in this group was higher than in the control groups. Although it was held for years that the part should be thawed slowly, recent work by Shumacker and Kunkler[148] has shown that rapid thawing in experimental animals may result in less gangrene than spontaneous thawing. Glenn et al.[149] have recently reported on the treatment of frostbite by adrenocorticotropic hormone, but they could find no significant therapeutic affect on severe cold injury.

Until the efficacy of other methods of treatment is proven, frostbite is treated at the Boston City Hospital by applying loose sterile dressings, administering chemotherapy and adopting a policy of cautious, watchful waiting.

Only dead tissue is excised! In most cases seen in civilian practice gangrene does not occur, and the effected superficial tissues will spontaneously peel away leaving relatively normal tissue underneath. Occasionally spastic phenomena may be present in such extremities for

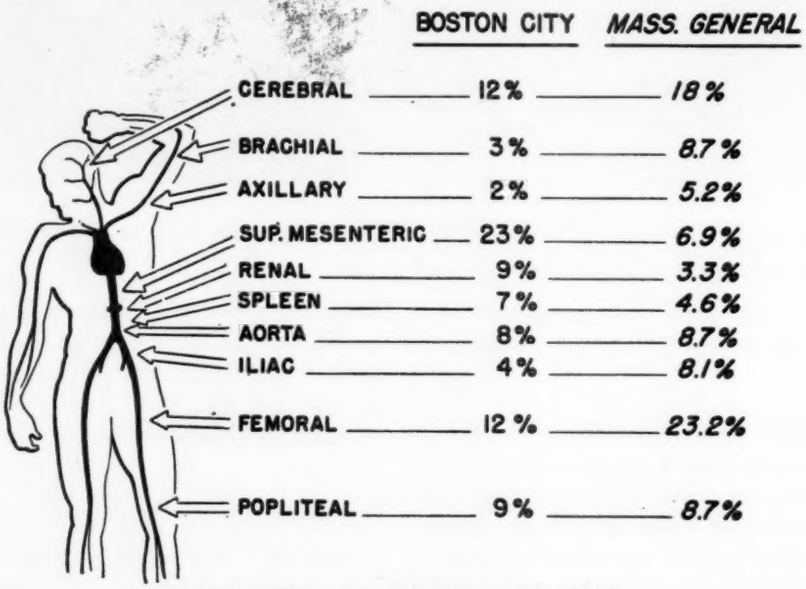

FIG. 33. Sites of peripheral arterial emboli.

months to years after the injury and may be relieved by sympathectomy.

OCCLUSIVE DISEASE

Arterial emboli lodge in the upper extremities in a variable percentage of cases. In a series of 172 arterial emboli cases reviewed at the Massachusetts General Hospital by Warren and Linton,[150] the upper extremity was involved in 13.9 per cent of the cases. In a series of 101 peripheral emboli cases reviewed at the Boston City Hospital, the upper extremity was involved in only 5 per cent of the cases. (Fig. 33.)

The source of arterial embolism is usually a diseased heart with auricular fibrillation. In a series of fifty-five cases seen at the Boston City Hospital the etiology was arteriosclerotic heart disease in twenty-six cases; rheumatic heart disease, seventeen cases; hypertensive heart disease, four cases; bacterial endocarditis, three cases. In four cases an arteriosclerotic plaque of the aorta predisposed to embolism. In one case there was a patent foramen ovale in which the source of the embolus was phlebitis of the right iliac vein.

Upper extremity emboli rarely result in gangrene. Conservative therapy should be followed consisting of sympathetic blocks and vasodilating drugs. Rarely, if there is underlying obliterative vascular disease, gangrene may occur as seen in Figure 34.

Buerger's disease rarely affects the upper extremity. Silbert[151] reported on a group of 1,400 patients with this disease, in which only five patients had major amputations of the upper extremity. Hamlin et al.[152] reviewed a series of 180 cases at the Massachusetts General Hospital and found that only 6 per cent required amputation of the fingers, with no amputation above the level of the finger being required. Treatment consists of sympathectomy, cessation of smoking and control of sepsis with antibiotics.

Obliterative arteriosclerosis results in gangrene in upper extremities in a very small percentage of cases. Treatment is usually conservative.

SPASTIC DISEASES

Raynaud's disease is characterized by a symmetric arterial spasm of the fingers when stimulated by cold or excitement. (Fig. 35.) Although it seldom goes on to gangrene, it may be extremely disabling. Treatment consists of administering vasodilator drugs such as roniacol® and priscol® to the mild cases and performing a sympathectomy on the more severe cases. Although the latter does not necessarily cure the disease, it gives great relief in many cases. This is especially true if there

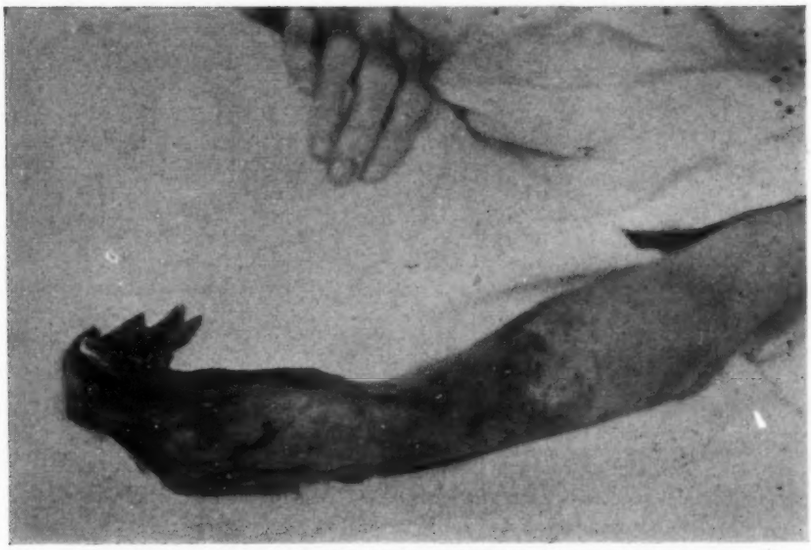

FIG. 34. Gangrene of hand resulting from embolus in arteriosclerotic extremity.

FIG. 35. Raynaud's disease demonstrating symmetric arterial spasm.

is marked coldness and sweating of the extremities between paroxysmal attacks.

Reflex vasospasm may be secondary to many conditions such as cervical ribs, scalenus anticus syndrome, coronary occlusion and trauma. Figure 36 demonstrates the right hand of a dentist who accidently punctured his thumb while injecting novocain solution. Infection occurred around the puncture site which disappeared in two weeks. Subsequently the hand became weak and cold, and necrosis of the tips of the thumb, index and middle fingers developed. Physical examination revealed absent pulses in the wrist and antecubital fossa. A blood pressure was unobtainable in the right arm. Treatment was instituted

FIG. 36. Reflex sympathetic dystrophy resulting in gangrene of tips of thumb, index and middle fingers in a dentist who injured right thumb at work.

37

38

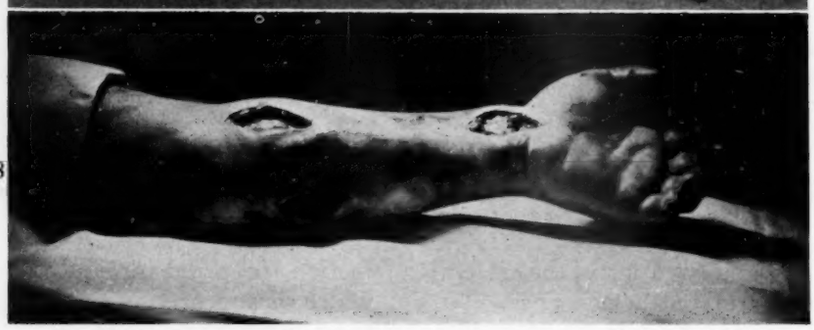

FIG. 37. Digital gangrene developing shortly after onset of virus pneumonia.

FIG. 38. Gangrene of upper extremity resulting from massive infection with hemolytic streptococcus.

twenty-three days after the initial injury and consisted of sympathetic blocks, priscol, roniacol and local treatment to the necrotic fingertips. Within several days a faint pulse became perceptible in the right wrist. The hand gradually improved, with the blood pressure in the right arm returning to 105/85 compared to the left arm of 140/85. The fingertips healed and there was no limitation of motion in the fingers or intermittent claudication in the forearm on resuming work.

INFECTION

Systemic infection may give rise to severe paroxysms with marked vasoconstrictor activity. It is conceivable that such severe vasoconstriction could damage the intima of the blood vessels with subsequent thrombosis. This may explain some of the cases of gangrene of the fingers which have resulted from influenza, virus pneumonias, etc. Figure 37 is the picture of an elderly man who developed acral gangrene approximately one week after the onset of virus pneumonia. This resulted in amputations of the portions of the finger which are demarcated in the photograph. No circulating agglutinins were found in this patient.

Infection localized in an extremity may result in gangrene. Figure 38 demonstrates such a case. This was a fifty-seven year old white morphine addict who injected heroin into his left arm near the elbow four days before entering the hospital. Infection developed and spread to the entire forearm. The hand and fingers were cyanotic, and no pulse could be felt in the wrist. Several incisions were made and creamy pus was obtained which cultured hemolytic streptococcus. On entry he was placed on penicillin therapy, but the circulation in the arm did not recover. Exploration of the antecubital fossa was performed which revealed the brachial, radial and ulnar arteries to be in intense spasm. Saline compresses over these vessels caused some pulsation to return. A periadventitial sympathectomy was performed, and at the conclusion of the procedure the blood vessels appeared to regain some pulsation. Numerous stellate blocks were performed but they did not appear to affect the situation. The hand and forearm progressed to gangrene and the patient required an emergency guillotine amputation of the forearm with a subsequent revision of the amputation stump. Examination of the amputated extremity revealed no thrombosis in the radial or ulnar arteries, which were found to be in intense spasm. One other factor which may have been of importance in the pathogenesis of this condition was the fact that all the superficial veins of the forearm were obliterated by thrombosis due to the numerous previous narcotic injections. It is well known that extremities involved with phlebitis are less tolerant of infection than normal ones.

VII. PAINFUL STATES OF THE HAND

There are several conditions which produce painful states of the upper extremity, and these should be carefully distinguished from each other.

Disuse of a part, with or without ischemia of soft tissues, will lead to limitation of motion of joints and atrophy of the soft tissues. Attempted use of such an extremity may result in pain. This type of pain is due to disuse atrophy and may be called *dystrophic pain*. Treatment consists of physiotherapy to restore motion to the affected part. One sees this more commonly in the elderly individual or in those individuals with a rheumatoid diathesis.

Amputation of a part almost universely results in some manifestation of the *phantom limb*. The affected person feels that the amputated part is present. Reassurance by the personnel involved in treating such a patient will do much to diminish any painful experiences which might be associated with such a sensation. In the great majority of cases the phantom limb is of no consequence; but rarely if there are emotional or psychiatric problems, such sensations may be contorted in the central nervous system to a definite painful phenomenon. Treatment of such cases should be along psychotherapeutic lines.

Amputation neuromas have been mentioned in a previous section. They may cause exquisite pain in an extremity and set up a reaction of pain along the central nervous system pathways, so that an entire extremity can become painful.

Such neuromas should be carefully sought for by examination of the wound, using a small palpating area such as the blunt point of a pencil. When there is definite evidence of a

localized spot in the scar that will cause severe pain and is associated with paresthesias, operation is indicated. At times the neuroma itself may be felt as a discrete mass. Correct surgery demands excision of the neuroma with implantation of the proximal nerve either in soft tissues or in bone. Blind excision of scar tissue is of no avail, since the neuroma will reinsert itself in the subsequent scar.

Such painful neuromas are often confused with the phantom limb syndrome since they may appear simultaneously with it. Sometimes excision of an amputation neuroma will relieve a painful phantom. This does not imply that the phantom limb syndrome is due to the painful neuroma. It is only that the painful neuroma so excites or stimulates certain areas of the central nervous system that the phantom sensation becomes painful. Routine wound revisions are of no value in the treatment of phantom limb. They are of value only when there is a definite painful amputation neuroma which should be treated as such.

Painful amputation neuromas should not be confused with causalgia. Pain produced by them should not be named "minor causalgia" since this only adds to the confusion. Sympathectomy is of great value in causalgia. It is of no value in the amputation neuroma syndrome. In fact, the dry skin produced by the operation further alters sensory patterns reaching the central nervous system and may increase the pain.

The spread of pain from the amputation neuroma to involve the entire extremity may be explained by the fiber interaction in injured nerves which has been studied by Granit et al.[153] and Rosenblueth.[154] Another theory is that the painful nerve impulses so stimulate or excite the internuncial pool of the spinal cord that normal impulses reaching this area from the periphery are interpreted as painful. Such could also occur in higher regions of the central nervous system.

A reflex may take place by way of the sympathetic nervous system following trauma, resulting in ischemia of the part. This was mentioned in the previous section on reflex arteriospasm. Such cases may be associated with a variable amount of pain due to ischemia and dystrophy and has been named *reflex sympathetic dystrophy*. Treatment of these cases is by sympathetic block and sympathectomy. If the patient's condition does not permit this, various drugs such as priscol and roniacol may be of value.

Causalgia is the definite clinical entity which may follow nerve injury. It is a spontaneous pain, hot and burning in character, aggravated by temperature changes, emotions, etc. The involved part is extremely sensitive to touch or even to the suggestion of being touched. It may lead to profound changes in the emotional state of the patient.

The cause of this painful state is basically unknown, although a short-circuiting between sympathetic and sensory nerves at the site of injury, as suggested by Doupe et al.,[155] explains many of the clinical characteristics.

Mayfield[57] observed 105 causalgia patients at the Percy Jones General Hospital during World War II. They comprised 5 per cent of all patients admitted to this hospital with peripheral nerve injuries. He confirmed the fact that sympathectomy brought complete relief of pain whenever it was completely performed. Sympathetic nerve block with procaine produced dramatic temporary relief of the causalgia, but in none of his cases did it give permanent relief even when repeated six to eight times.

Shumacker et al.,[156] in a review of ninety cases of causalgia seen at the Mayo General Hospital in World War II, had a somewhat different experience. They found that in twenty-one of eighty-three patients in whom one or more sympathetic nerve blocks were performed, permanent relief of symptoms was obtained.

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A GOITER in the posterior mediastinum is very rare, but invariably it is derived from a cervical goiter even when connective fibers are no longer present. Such a "false accessory goiter" usually lies directly in front of the spinal column and especially to its right side because of interference to its growth to the left that is offered by the arch of the aorta. The superior vena cava and ascending aorta are invariably in front of it and almost always the trachea and esophagus also lie anteriorly. Hence the best approach to this growth is the transthoracic one. (*Richard A. Leonardo, M.D.*)

An Improved Postoperative Adhesive Dressing

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POSTOPERATIVE abdominal dressings are usually large and bulky and tend to restrict diaphragmatic motion. The removal and reapplication of adhesive is attended with discomfort to the patient, and therefore inspection of the wound is often deferred. Corset type dressings with the free edges being approximated by applicator sticks and rubber band,[1] zippers,[2] etc., remove some of these objections, but they are still bulky and restrictive. Zippers, etc., have no advantage over ordinary gauze ties from the standpoint of simplicity and economy. A simple abdominal dressing will be described that satisfies most of the criteria to be sought for in a protective supportive postoperative dressing.

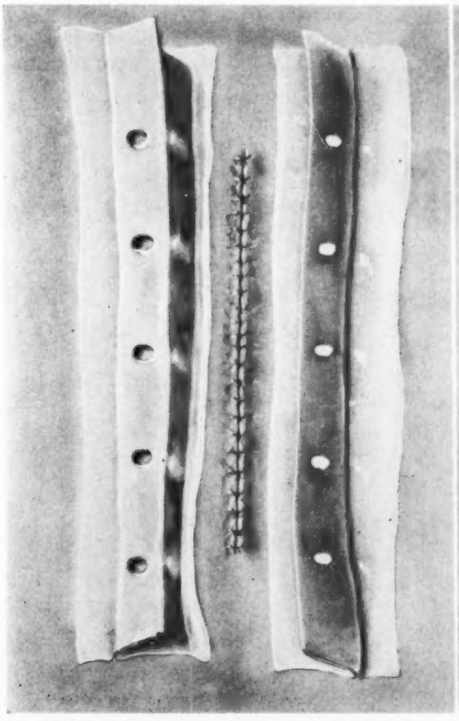

FIG. 1. Adhesive tape, previously prepared, is placed parallel to the incision.

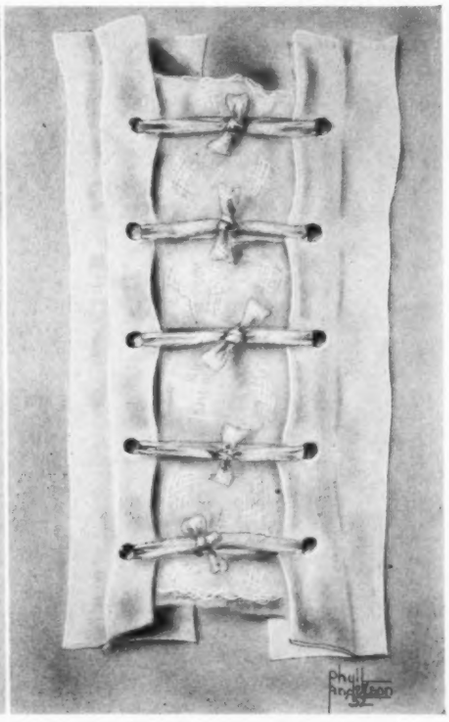

FIG. 2. Gauze ties exert traction on the tape and keep the dressing in place.

Two strips of either 3-inch elastoplast* or adhesive are cut to extend an inch longer than the incision, in order to distribute the pull and prevent the edges from becoming loose. The adhesive is then folded in the center, leaving a margin on both sides. Preparation may be

* Elastoplast manufactured by Duke Laboratories, Inc., Stamford, Conn.

facilitated by placing the adhesive on two pieces of plywood that are separated about 1 inch. The plywood is then apposed and the adhesive is folded. Holes are made in the folded portion with an ordinary puncher. Ace adherent* or tincture of benzoin is applied to the skin. These agents increase the adhesive power and tend to diminish any irritative effects of the adhesive. The adhesive is then placed on each side and parallel to the incision. (Fig. 1.) A gauze dressing is applied and the tape is drawn together with cross ties of gauze or other material. (Fig. 2.)

The advantages of this dressing may be listed as follows:

1. The dressing is easily prepared, and is time-saving and inexpensive.
2. Frequent changes of tape are unnecessary.
3. Inspection of the wound and change of dressing is easily made.
4. Wound-healing is aided by relief of tension and relaxation on the line of incision.
5. The dressing is not bulky and occupies a relatively small area of the abdomen. The tendency toward postoperative pneumonia and atelectasis is diminished since freer abdominal motion is allowed.
6. It is also useful where draining wounds are present.
7. Perforations in the tape may be used to anchor drainage tubes.
8. By altering the size of the tape this principle may be utilized in small incisions, may aid in the approximation of minor lacerations and may be used to approximate wounds healing by secondary intention.

* Ace Adherent manufactured by Becton, Dickinson and Co., Rutherford, N. J.

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External Fixation of Nasointestinal Tubes

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A METHOD is presented that provides satisfactory stationary fixation of gastrointestinal tubes to the nose. For the most part it is not possible to eliminate the uneasiness associated with prolonged intestinal intubation, but this discomfort is unnecessarily aggravated when bizarre forms of adhesive fixation extend over the face, the malar eminences, the forehead and the periorbital area. Insecure fixation may be accompanied by alteration in the position of the tube after it has been properly placed. It may be impossible to reestablish this position when a tube has been placed in relation to a suture line or into a single loop of intestine for decompressive or feeding purposes. This method has been found more satisfactory than suture fixation to the alae nasi.

The fixation described is not original and is an adaptation of a commonly used method of fixing urethral catheters in place.

After the tube has been properly positioned and dried, the bridge of the nose and the alae nasi are dried and painted with tincture of benzoin. A 4 by ½ inch strip of adhesive is divided in half so as to form two 3 inch long legs. The broad base is secured smoothly to the nose between the bridge and the alae. Each leg is whip-wrapped about the tube, one starting clockwise and the other counterclockwise. (Fig. 1A.) The base of the strip is further fixed by one or two ½ inch wide adhesive strips applied across the short axis of the nose. (Fig. 1B.)

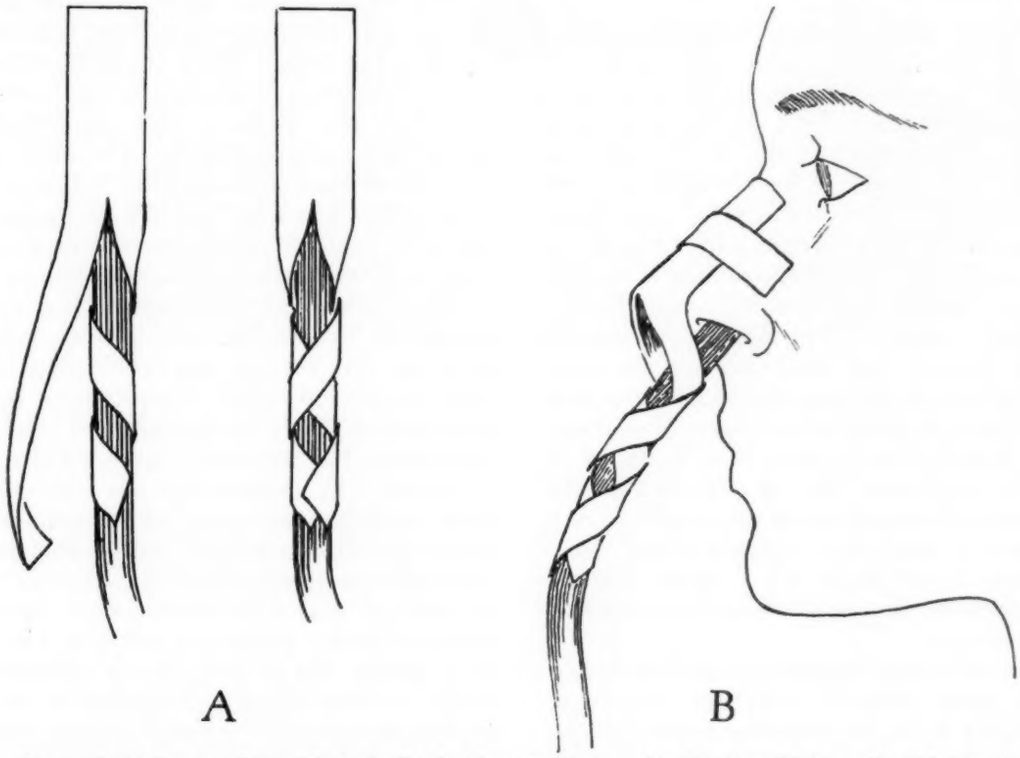

FIG. 1. A, illustrates the method of adhesive fixation of the tube; B, the adhesive-whipped tube is fixed transversely to the nose.

Local Anesthesia in Pediatric Surgery with a Description of an Approach to Gain the Child's Acceptance

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A SURGEON performing an operative procedure on a child usually uses a general anesthetic in cases where he would often use a local anesthetic agent for a similar problem in an adult. This frequently makes an ambulant procedure one which can be accomplished only in a hospital. The problem of emotional trauma surrounding surgery in children is obvious and has been discussed by Bakwin,[1] Coleman,[2] Kaplan and Jessner,[3] Levy,[4] Eckenhoff[5] and others. The Boston Children's Medical Center has prepared an excellent pamphlet[6] which discusses the honest approach to a child about to undergo surgery. Leigh and Belton[7] have pointed out in their textbook of pediatric anesthesia that 10 per cent of the anesthetics administered by them were spinal. Farr[8] and Evans[9] have written about local anesthesia in children. This paper will be concerned with the use of topical and infiltration anesthesia.

The use of a local anesthetic agent in a child depends upon three factors: (1) pathology, and whether it can be eradicated with the use of such an anesthetic agent; (2) the child; (3) the surgeon. A deep-seated or widespread pathologic process is obviously not a suitable situation wherein local anesthesia can be used. A cooperative child can be evaluated by the surgeon on seeing the youngster during his preoperative examination as well as by discussing his reactions with the parents and family physician. The surgeon himself should be used to working under local anesthesia, should recognize the need for gentle handling of tissues, and be of such temperament that he will not be disturbed by the sometimes electric atmosphere under such conditions.

TECHNIC

This method relies upon full understanding by the child of what is to be done, step by step, explained in simple words of gentle encouragement. Preoperative sedation is often not necessary and sometimes, in fact, may make the procedure impossible to do. The child is told first about the coolness of the antiseptic solution which is to be applied to prepare the operative site. No drapes are placed about the area; a towel can be applied to the lower angle of the site so that the surgeon may rest and steady his hands. The child is then told that he or she will feel only one needle pinprick, the first intradermal wheal. The anesthetic solution is deposited slowly in the skin and subcutaneous tissues. The transitory burning and stinging is foretold (the surgeon who has been operated upon himself under local anesthesia can best appreciate the sensation). Subsequent infiltration with the needle should start always within an area of blanched, anesthetized dermis so that no further punctures are felt. The child may watch this if it is on an extremity. A needle is then taken and applied to the area to show that pain is no longer present and that anesthesia has developed. When the child accepts this fact, the operative site is repainted and the drapes applied. The surgery then can be carried out with ease. At no time should one go beyond the anesthetized area because this will make the child apprehensive. The surgeon should remember always that a much wider area of cutaneous infiltration is needed for the suturing process than for the initial incision and he should deposit sufficient local agent to anesthetize the areas where the needle points will occur when closure is effected.

This technic has been used in children as young as two years of age. Although first applied to the extremities, it has been useful

in operations on the head and neck and trunk as well.

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Thymic tumors are very rare, and five of six cases are lymphosarcomas or carcinomas. Benign tumors can be removed by a transthoracic approach with partial or total extirpation of the sternum, but malignant tumors often present metastases and are inoperable. Simple hyperplasia of this gland is frequently found in patients with myasthenia gravis but surgical extirpation of the thymus produces only temporary improvement, according to recent statistics. (*Richard A. Leonardo, M.D.*)

The Heineke-Mikulicz Principle in Surgery of the Anus and Rectum

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THE Heineke-Mikulicz principle is one that has been utilized in operative surgery for many decades.[1] It has a field of application that is extremely wide, being employed in several fields of surgery, especially of the anus and rectum. The indication for its use is in the correction of a stenosed ostium or in the primary repair of a lumen in such a manner as to prevent stricture formation. The simplicity of the procedure does not detract from its value for it has amply stood the test of time, even in these recent years of rapid progress in surgical technic.

The basic principle is the longitudinal incision of a stenosed area with closure in the transverse plane, or the longitudinal excision of tissue with transverse repair if stenosis is to be avoided. This principle has a wide use in anal surgery. It is employed in the operation for the cure of fissure in ano or anal ulcer. In this instance the fissure is widely excised in an elliptic manner and the mucosal defect is approximated to the superficial fibers of the external sphincter muscle in a transverse direction. This procedure is a direct application of the Heineke-Mikulicz principle.

One of the most efficient surgical methods to correct stenosis of the anal canal, especially following a hemorrhoidectomy, is the procedure described by Martin.[2] In this operation an incision is made in the skin and carried through the stenotic ring as far as the anorectal line. A gaping wound is the result. The mucosa is freed by sharp dissection for a distance of 1 cm. and fixed with interrupted catgut sutures to the subcutaneous tissue at the anal margin in a transverse manner. This transverse wound, when completed, will be 1.5 to 2 cm.; thus another demonstration of the Heineke-Mikulicz principle is effected.

The radical or amputative hemorrhoidectomy described by Buie is a procedure employed for extensive hemorrhoids, especially when accompanied by prolapse of the rectal mucosa. This entails the removal of the hemorrhoidal area for the greater portion or for the entire anal circumference. If it is performed for the entire anal circumference, it is usually done in three segments. Each segment is removed in the longitudinal axis of the anal canal and the resulting defect is closed by approximating the edges of the anal mucosa to the superficial fibers of the external sphincter muscle in a transverse manner. Many proctologists employ this type of closure in a routine hemorrhoidectomy which allows an area of mucosa and skin to remain between each excised segment that is larger than that left in the amputative type of removal.

Lockhart-Mummery[3] has described a procedure for the correction of a rectal stricture which is the exact principle under discussion. A longitudinal incision is made through the rectal wall in the stricture area and then repaired in a transverse manner.

Another useful application is in the end-to-end anastomosis of the rectum and sigmoid after anterior resection has been performed. If it appears that the os may be too narrow, each segment of bowel may be incised along the antimesenteric border for a centimeter or more before the repair is made. This will allow for a wider suture line and a larger os.

I am sure that I have not evaluated all of the applications of the Heineke-Mikulicz principle in proctologic surgery. I readily grant that there is nothing new in the Heineke-Mikulicz procedure, but it is of such great value that it is worth while to focus attention upon a truly fine and basic surgical maneuver.

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Misleading Position in Sigmoidoscopy

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FOR the past two years I have been using the so-called Ritter proctologic table and have found it to be of great aid in sigmoidoscoping patients who otherwise might not be cooperative or comfortable. Recently I had an experience with this table in which its practical use almost worked against its own advantage.

On January 29, 1954, J. I., a sixty-four year old man, presented himself with rectal bleeding of two days' duration. Digital examination was essentially negative. Proctoscopic examination revealed large prolapsing internal hemorrhoids which were obviously the source of his bleeding.

As was my custom, I sigmoidoscoped the patient purely as a precautionary measure. The patient was placed on the table which was tilted to its full turn; this placed the patient at approximately a 60 degree angle. An 11 by $\frac{5}{8}$ inch sigmoidoscope was passed with ease for its full distance. As the sigmoidoscope reached "bottom," for a fleeting second a spot of red was seen. I could not be sure that I had not imagined this or that I had not seen a reflex of light, but I was disturbed. Removing the sigmoidoscope, I again passed it for the full distance. At this time I saw nothing whatsoever. The sigmoidoscope was removed and I tried again with a 10 by $\frac{3}{4}$ inch sigmoidoscope, believing that if one could dilate the bowel a bit it could foreshorten the bowel. Again nothing was found. I should probably add that it is not my custom to use air inflation during sigmoidoscopy.

The table was straightened; the patient descended and was made to jump up and down several times. He was placed back on the table, which was tilted 5 degrees and raised to its greatest height, and the sigmoidoscope was passed once more. Under these conditions a tremendous polypoid mass was picked up at approximately the 6 inch level. It completely filled the mouth of the sigmoidoscope. I carried it the full length of the instrument, still carrying the tumor, and was able to push it out of sight with the aid of a cotton wiper.

I had just proved to myself that I had a polyp on a long pedicle which was attached a distance above the 11 inch marker from the anus. Had the table not been tilted 5 degrees and had the patient not hopped up and down, the polyp would have been easily missed.

The patient entered the hospital where a barium enema was given and a second polyp was found above the first. On February 15, 1954, simple colotomy was performed and both polyps were removed. The lowermost polyp, which was the one in which I was most interested, surprisingly enough had a pedicle of only 3 inches. Theoretically, this polyp should have been at least 6 to 7 inches long since it was picked up at the 6 inch level and carried to the 11 inch level. This makes 5 inches of sight and at least 1 inch beyond, or more.

We can explain this phenomenon by the fact that in the second position the patient was intussuscepting one side of the bowel which in itself acted like a pedicle.

CONCLUSIONS

The completely "upside down position," which is usually employed in examining patients on a Ritter table or a table of the same type, is a great aid in examination. At the same time, if a polyp has a high attachment and great mobility, it may be allowed by gravity to fall out of sight and the diagnosis will thus be missed.

Massive Metallic Implant *in Situ* Five Years after Resection of Mandible

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THE cosmetic liability which follows hemiresection of the mandible is not great. This is true whether or not the resection is performed in association with radical neck dissection providing that *more* than a half of the mental process is kept intact.

If the entire mental process must be sacrificed, the cosmetic liability increased proportionately as the amount of contralateral jaw removed increases. In such cases the use of a prosthetic device should be seriously considered. Such prosthetic devices can be accurately planned and fashioned preoperatively. If cases are properly selected and if scrupulously correct technic is employed, there is no reason why such an implant should not remain in place for many years. (However, few cases of malignancy involving the mandible are suitable for this procedure.)

Intensive precision preoperative x-irradiation therapy has been delivered to the mandible and future site of metallic implant, then followed by mandibular resection and immediate prosthetic repair of the defect with complete success. It must be emphasized again, however, that few cases of malignancy of the jaw can be selected for this type of management. Full correlation of radiologic, surgical and oral prosthetic knowledge is vital to success.[2]

Because of the relatively meager surgical literature dealing with the subject, this contribution is offered.[1] In the case reported herein x-irradiation therapy was employed for an advanced primary intrinsic malignancy of the jaw with disruption of the tumor into the oral cavity proper. The case was at first considered inoperable. The lesion was modified by intensive irradiation therapy to such an extent that the case was re-evaluated and pronounced technically and categorically operable.[3]

CASE REPORT

A white man, thirty-eight years of age, was first referred to me in April, 1948, with a history of swelling involving the left side of the mandible. Because of the swelling and pain in the region of the mandibular left cuspid tooth, the patient had visited his dentist in November, 1947. At that time the offending tooth was removed, but the pain was not relieved. Shortly thereafter, the gingival tissue became "swollen." Two weeks later the patient visited a second dentist who incised the area, releasing purulent material.

Four months after the onset of symptoms, because of continuing pain and increasing swelling, the patient consulted a third dentist. The remaining mandibular anterior teeth were removed and the area of most prominent swelling was again incised, without drainage. Because of the history and unusual conditions noted, the dentist obtained a biopsy specimen from the piled-up and indurated tissue springing from the socket of the previously removed mandibular left cuspid tooth. A diagnosis of probable malignant tumor was made from the biopsy.

The past medical history was essentially irrelevant to the present condition. There was no family history of cancer.

In April, 1948, when the patient was first examined in this office, there were no pertinent or significant abnormal physical conditions other than the local lesion.

The temperature, pulse, respiration and blood pressure were within normal limits.

The maxilla and mandible were edentulous. The patient was able to open and close his mouth without difficulty. An expanding mass presented on the lingual surface of the mandible which extended from a point just left of the symphysis to the left molar region. This mass bulged the lingual plate about 1.5 cm. The labiobuccal plate was also expanded from the symphysis to the left bicuspid region. The alveolar process was about 0.5 cm. above its normal level. Upon palpation of the mass the egg-shell sign could not be elicited. On the left outer surface of the alveolar process of the mandible an ulceration with fungating edges could be seen which extended from the region of the left lateral incisor to the region of the left first molar. This apparent neoplastic ulceration did not infiltrate into the sulcus. It represented an obvious neoplastic breakthrough or eruption of the buccal plate.

Examination of the cervical regions revealed no evidence of malignant lymphadenopathy.

In the occlusal roentgenograms, cortical expansion of the osseous structure was obvious. Fine radiating spicules perpendicular to the surface of the involved bone were observed. These radiating forms of periosteal ossification were suggestive of subperiosteal neoplastic infiltration. A roentgen survey included studies of the long bones, skull and chest. No evidence of osseous or pulmonary metastasis was noted.

Although a diagnosis of probable malignant tumor was made from the biopsy specimen obtained from the outside institution, the slides were considered inadequate for unequivocal diagnosis. Because of this a second biopsy was performed and a diagnosis of anaplastic malignant neoplasm of the mandible was made.

External irradiation therapy was delivered to the tumor of the jaw through a carefully controlled area, measuring 11 by 6 cm. A special lead mask was constructed to protect normal structures.[4] A total of 3,500 r (in air) was delivered to this area, using the following factors: 200 kv.p.; 50 cm. target skin distance; 1 mm. copper plus 1 mm. aluminum filtration; 300 r (in air) per dose from April 26 to June 9, 1948.

Peroral irradiation therapy was delivered to a carefully controlled area, using a special lead cone with a 3.5 by 4.8 cm. opening on a 5 cm. master cone attached to a Castigliano periscope.[5,6] A total of 8,100 r (in air) was delivered, using the following factors: 200 kv.p.; 40 cm. target tumor distance; 0.5 mm. copper plus 1 mm. aluminum filtration; 300 r (in air) per dose from April 27th to July 1st.

The total dose was 11,600 roentgens at 2 cm. depth. A severe irradiation mucositis and moderate epidermitis developed during ther-

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FIG. 1. Vitallium prosthesis which was planned and developed employing roentgenographic and topographic measurements as guides. The prosthesis when actually used in the patient was modified so that in cross-section the appearance was that of an angle iron rather than "U" shaped. (Courtesy of *J. Oral Surg.*[1])

FIG. 2. Prosthesis attached to a dry model representing the remaining fragment. Note the three screws employed for anchorage instead of the usual two. (See text.) (Courtesy of *J. Oral Surg.*[1])

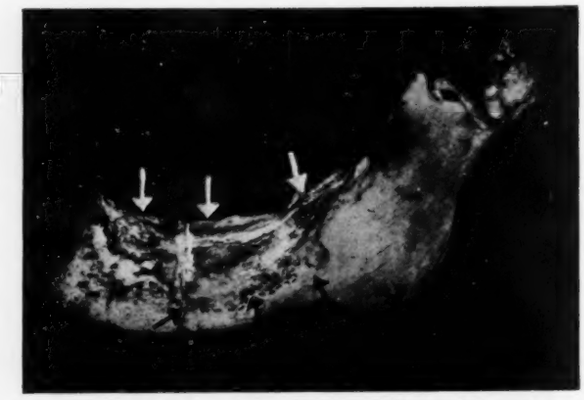

FIG. 3. Resected mandible. Dotted lines indicate limit of attached soft tissue lingually. Arrows point to floor of mouth; white arrows point to buccal surface. (Courtesy of *J. Oral Surg.*[1])

apy, but had completely subsided by August 6th. Complete healing of the break-through of the buccal plate and of the site of the original lesion was noted. Some expansion of the lingual and buccal plates was still evident.

In view of the marked reduction in tumor size and the healing of ulceration the lesion was now considered to be operable.

It was concluded that the patient should be given the benefit of radical surgery in order to give him the best possible chance for long-term survival. Repeat roentgenographs of the chest, long bones and skull revealed no evidence of metastasis. The neck was negative for metastatic nodes. In view of these observations resection with immediate prosthesis was planned.

A Vitallium® prosthesis was constructed from external and roentgenographic measurements of the patient's mandible. (Figs. 1 and 2.) A plaster head cap was constructed on a felt base, and on each side of the head cap Roger-Anderson screw fixation and bar attachments were inserted. These were firmly embedded in the plaster. The bar from the right side of the mandible was later attached to this cap to maintain rigid support of the mandibular fragment and the prosthesis.

On November 24, 1948, under local anesthesia, three Bigelow screws were inserted in the body of the mandible anterior to the angle and above the mandibular canal. A Bigelow bar was temporarily inserted to keep the screws in position for the next stage of the procedure.

The entire operative procedure was accomplished under local anesthesia as follows: bilateral injection of 4 per cent procaine into the third division of the trigeminal nerve in the foramen ovale at the base of the skull; nerve block of the second and third cervical nerves with 2 per cent procaine and infiltration of 1 per cent procaine into the soft tissues of the neck. Transient severe headache followed injection at foramen ovale.

The entire operation was performed intraorally. Sutures were placed in the tip and left mid-tongue for traction and exposure. An incision was made, starting in the retromolar region of the right (contralateral) side along the crest of the ridge to the right cuspid region. The incision at this point was directed downward past the sulcus and in the buccal surface, then continued parallel to and about 1.5 cm. below the crest of the ridge to the left retromolar region. At this point the incision was carried up to the crest of the ridge and extended to the tip of the coronoid process. The same procedure was then performed on the lingual surface with the incision deep in the floor of the mouth. The incision everywhere was kept wide of the involved region.

After completion of the incision careful hemostasis was established. A curved hemostat was introduced under the mandible in the right cuspid region. A Gigli's saw was brought under the mandible, cutting the mandible at that site. The mucoperiosteum beyond the involved portion was retracted and the mandibular muscle attachments were cut wide of their points of insertion in the section of the mandible to be removed. The coronoid process was cut away from the mandible with a bone-cutting forceps and the left hemi-mandible was rocked out of the glenoid fossa. The fact that the incision was made wide of the involved portion of the mandible permitted the gingival tissues, mucoperiosteum and muscles attached to the irradiated region to remain intact over the bone. The entire mass was then removed with the resected mandible. (Fig. 3.)

Cutting the coronoid process is important. The rocking, twisting and complete turning of the mandible, which is so often necessary to bring about the separation of its attachments, is a very traumatizing procedure with the coronoid process left intact. With it cut, the plowing action of the coronoid process is eliminated and the mandible can be twisted on itself without fear of trauma. This technic, developed by surgeons many years ago, but

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FIG. 4. Prosthesis in position at operation. Muscles of the floor of the mouth were sutured to the crib of the prosthesis, with sutures tied on the lingual surface. (Courtesy of *J. Oral Surg.*[1])

FIG. 5. Fixation of right fragment of mandible from Bigelow screws to the head cap by means of a Bigelow bar bent on itself and brought over the vertex of the skull and attached to the opposite side. (Courtesy of *J. Oral Surg.*[1])

little heard of, was essential for the successful performance of the operation. After removal of the mandible the coronoid process is easily removed. However, it is doubtful that its removal is necessary.

The Vitallium prosthesis was immediately inserted in the glenoid fossa and fixed to the mesial aspect of the right remaining fragment by three small Vitallium screws. The muscles of the floor of the mouth, including the genioglossus and the mylohyoid, were sutured to the crib of the prosthesis, with sutures tied on the lingual surface. (Fig. 4.) When actually used the prosthesis was modified from the appearance as seen in Figure 1 which shows channel iron effect in cross-section. The prosthesis was modified to that of an angle iron in cross-section.

This procedure was accomplished with the dual purpose of giving function to the muscles and providing elimination of the dead space.

The lingual and buccal tissues were then coapted over the prosthesis in such a way as to assure that the suture line would not be placed on the sharp edge of the implant.

For fixation of the remaining right mandibular fragment to which the prosthesis was attached, a 30 inch Bigelow bar was inserted in the slots of the preoperatively inserted Bigelow screws; one end extended upward and was attached to the head cap anteriorly. The other end was bent on itself outward and was attached to the right side of the head cap. The bar was then bent over the vertex of the head and attached to the opposite side exerting outward pressure on the jaw. This technic was employed to prevent both vertical and lateral motion of the fragment. (Fig. 5.) Immobilization was continued for a period of ten weeks. The patient was hospitalized for two weeks after the operation.

The following medication was used preoperatively and postoperatively until discharge: penicillin, 900,000 units daily for two days prior to the insertion of the Bigelow screws and continuing after discharge from the hospital; streptomycin, 0.25 gm. every six hours, one day prior to resection and continuing for eleven days postoperatively; sulfadiazine, 15 gr. every four hours, second day postoperatively and continuing for nine days; parenteral vitamin therapy, ascorbic acid, 600 mg. daily for eleven days postoperatively, then 200 mg. daily, orally for remainder of hospital stay; vitamin B complex for fourteen days.

After discharge, the patient was given repository penicillin, 4 cc. every three to four days, and supplementary vitamins (A, B and C) by mouth for two and a half months.

The final pathologic diagnosis was anaplastic primary malignant neoplasm of the mandible.

Follow-up studies were made December 10, 1953, sixty months postoperatively, at which time there was no evidence of recurrence or

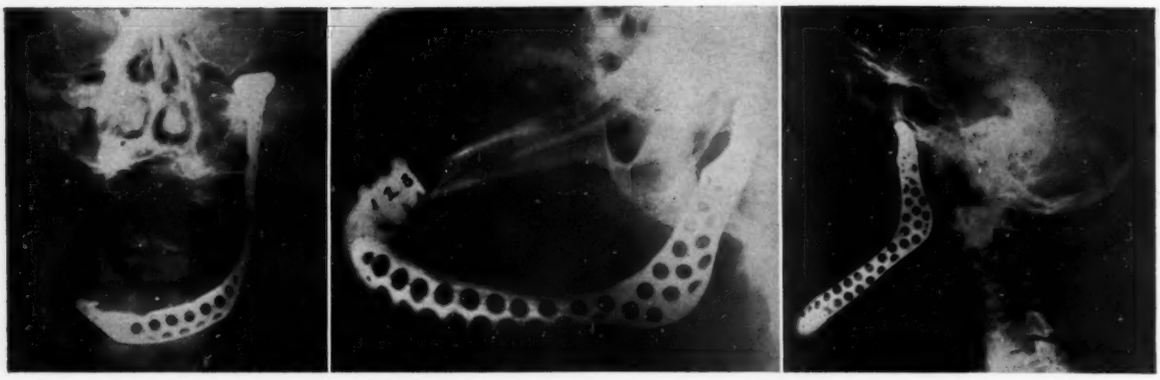

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FIG. 6. Roentgenogram taken sixty months postoperatively. Note position of condyle and attachment of the prosthesis to the right mandibular fragment. Stable union persists without evidence of necrosis.

FIG. 7. Lateral oblique view of prosthesis demonstrating union with right fragment of mandible. Note the three screws (1, 2 and 3) still solidly in place sixty-two months postoperatively.

FIG. 8. True lateral view at sixty-two months.

9A 9B

FIG. 9. A and B, sixty months after resection of the mandible there is little apparent cosmetic liability; function is adequate.

development of metastatic disease. Roentgen studies sixty months postoperatively indicate good position and stabilization at the site of junction of the prosthesis to the right mandibular fragment. (Figs. 6 to 8.) Mobility of the mandible and function are adequate and there is little facial deformity. (Figs. 9A and B.)

On February 23, 1951, the patient had partial gastrectomy performed because of gastric ulcer.

COMMENTS

Irradiation therapy is generally believed to be of little value in the treatment of advanced primary malignant lesions involving the mandible except as a palliative gesture. At the American Oncologic Hospital intensive irradiation therapy has been used preoperatively in the treatment of certain primary malignant neoplastic bone lesions for the past ten years, with the idea of attempting complete tumor devitalization. Occasionally, this technic is applicable to lesions involving the mandible. Complete devitalization of the tumor is rarely possible. However, in a reasonable percentage of cases, a profound effect of irradiation therapy on the tumor can be demonstrated.

In the management of certain primary malignant neoplasms of the mandible the outcome is dependent upon the stage at which treatment is instituted, the histologic character of the lesion and the aggressiveness of the surgical procedure.

This paper presents an approach to the management of certain malignant neoplasms of bone. Several features offer a new approach to an old problem. The use of intensive preoperative irradiation therapy, carefully planned and executed with precision, is shown to be compatible with a radical surgical procedure. Furthermore, immediate massive prosthetic replacement of a full hemi-mandible is compatible with intensive preoperative irradiation therapy, providing the surgical procedure is not too long delayed and the technical details of irradiation rigidly controlled.

The recognition of the compatibility of intensive irradiation therapy and radical surgery brings within the scope of radical but nonmutilating surgery at least a few cases now considered categorically inoperable. The conversion of an inoperable case to the status of operability, although seldom possible, is worthy of more frequent trial. Although many primary malignant neoplasms of bone are not radiosensitive, the use of such intensive irradiation therapy delivered to a carefully controlled volume of tissue may exert highly desirable effects.

It is likely that tumor devitalization brought about by such irradiation serves a twofold purpose: (1) It may decrease the incidence of *preoperative* showers of tumor emboli, thereby decreasing the possibility of metastases. (2) It may decrease the incidence of successful tumor dissemination *during* the operative procedure. The profound irradiation effect on the tumor renders the residual cells, both qualitatively and quantitatively, less likely to set up foci of metastatis.

In addition, the use of preoperative irradiation in these advanced malignant neoplasms provides a reasonable period of observation. During this period of observation metastasis if present in subclinical form may develop sufficiently so as to positively declare itself, thus obviating useless mutilating surgery.

SUMMARY

A case of advanced primary malignant neoplasm of the mandible without evidence of metastatis was treated with intensive preoperative irradiation therapy (11,600 r),* followed by complete hemi-resection of the mandible by the oral route. An immediate prosthetic replacement which included a condylar process restoration was successfully accomplished.

The most recent roentgenographic and laboratory studies (sixty months postoperatively) of the case here presented show no evidence of recurrence or metastasis. Good callus formation developed and firm stabilization at the site of the junction of the prosthesis to the right mandibular fragment has been maintained. Mobility of the mandible and function of the condyle of the prosthesis are adequate. The deformity of the facial features is insufficient to be classed as a cosmetic liability, as can be judged by photograph of patient taken five years after operation. (Fig. 9.)

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* Actual tumor dose.

Complete Heart Block Complicating Pulmonary Valvulotomy

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THE following case is reported because of the unusual complication of refractory complete heart block following an apparently successful valvulotomy for congenital stenosis of the pulmonary valve. The authors were unable to find any similar case in the literature and therefore considered it to be of sufficient importance to be brought to the attention of physicians treating cardiovascular disease and, in particular, of those dealing with the surgical approach to these conditions.

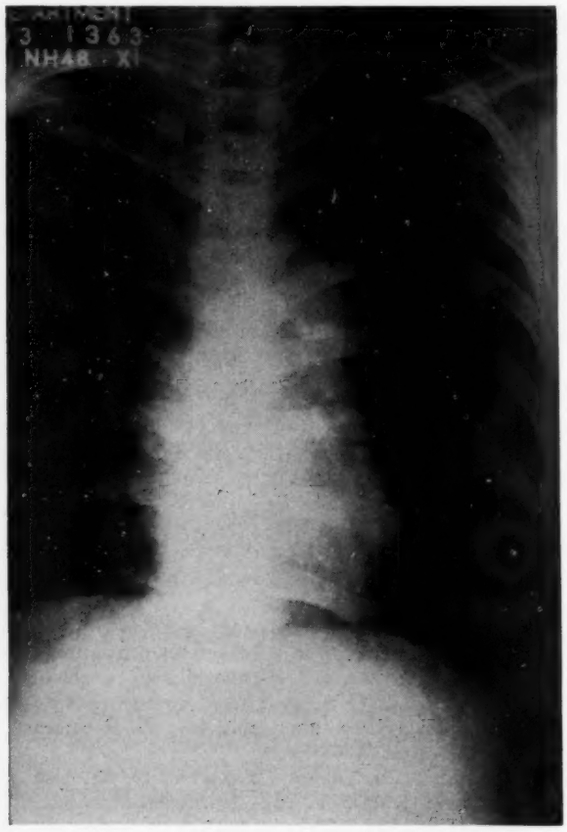

FIG. 1. Roentgenogram of the chest showing some enlargement of the right ventricle and marked accentuation of the main pulmonary arterial system.

CASE REPORT

The patient, a seventeen year old white girl, was hospitalized because of a congenital cardiac lesion which resulted in her complaint of dyspnea on exertion. Dyspnea was produced by effort equal to the ascent of ten stair steps. Fatigue and cyanosis accompanied the exertional dyspnea. There was a history of a "heart murmur" since birth. The patient's activity had been markedly restricted by the exertional dyspnea during her childhood. During the two or three years prior to admission to the hospital, the symptoms had increased in severity. Headaches and vomiting had occasionally been associated with chest pain. Recently there had been three episodes resembling petit mal seizures.

On physical examination there was cyanosis of the fingers and toes. A loud grade III, high-pitched systolic murmur was present over the precordium, with maximal intensity in the left second interspace. The murmur was projected to the left shoulder region and was well heard in the interscapular space. A systolic thrill was noted in the second left interspace.

Electrocardiogram showed right ventricular hypertrophy and sinus tachycardia. A systolic murmur was recorded on the tracing. Roentgenograms of the chest (Fig. 1) and cardiac fluoroscopy showed some enlargement of the right ventricle and marked accentuation of the main pulmonary arterial system. There was marked hypoplasia of the aorta. The peripheral vascular markings were considered within normal limits. An electroencephalogram was normal.

Cardiac catheterization showed marked pulmonary stenosis, probably of the valvular type, with right ventricular hypertension. The rise in

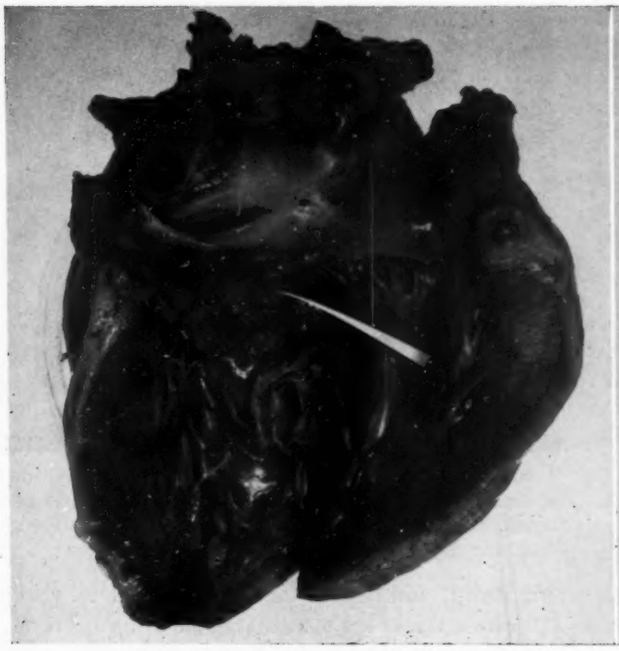

FIG. 2. Right side of the heart showing striking hypertrophy of both chambers. The tricuspid leaflets have been removed and the arrow points to the area that was traumatized.

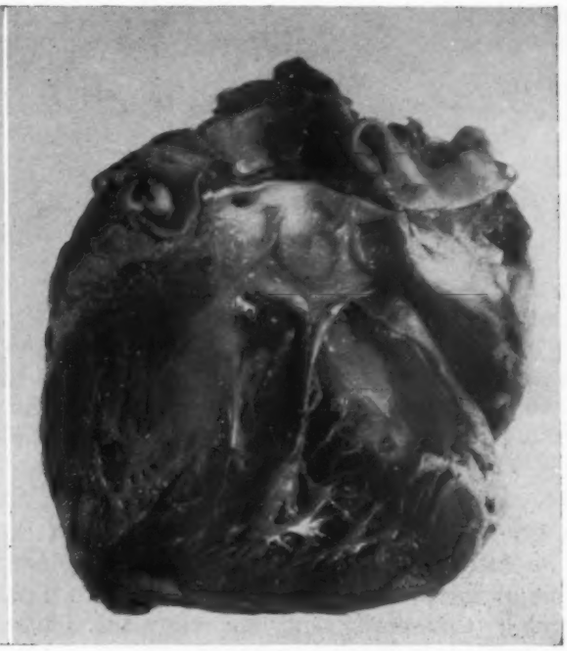

FIG. 3. Left side of the heart showing the area of subendocardial hemorrhage just beneath the left posterior cusp of the aortic valve. Note the dome-shaped pulmonic valve and its central perforation. It has been slit surgically almost to the edges of the valve attachments.

right atrial oxygen content in comparison with the vena cava samples suggested the presence of an atrial septal defect.

After the diagnosis of pulmonary stenosis had been confirmed a valvulotomy was performed through a small incision in the anterior aspect of the right ventricle, using the Gerbode valvulotome. The pulmonary artery pressure became elevated immediately after division of the valve, as indicated by increased turgor of this vessel. A marked thrill, which had been present over the pulmonary artery, disappeared.

A dilator was then inserted through the incision in the right ventricle. Difficulty was experienced in passing it through the wall of the ventricle and it was withdrawn. At this moment the patient's heart went into arrest. It resumed activity spontaneously within nine seconds, as shown on an electrocardiographic tracing which was taken continuously during the cardiac phase of the operation. Upon resumption of activity by the heart, a complete dissociation of A-V rhythm was noted. There was no change in the heart block after a period of delay, so it was decided to reinsert the dilator and complete the operation quickly. The dilator was passed through the sectioned pulmonic valve and opened. It was then withdrawn and the operation concluded. At this time, a thrill of low intensity was palpable over the anterior portion of the heart which had not been present previously.

Following operation the patient continued to have a two to one heart block. She became cyanotic and dyspneic. The administration of oxygen seemed to bring little relief. A loud blowing systolic murmur, grade IV, was present, maximal in the second left interspace. The pulmonic second sound was audible. It had not been so prior to operation. Arterial hypotension developed which was corrected by the use of levofed® solution in a continuous intravenous drip. The heart block continued despite attempts to convert the rhythm to normal by accelerating the rate. On one occasion for a period of a few minutes the heart converted to normal sinus rhythm but relapsed into two to one block. On the third postoperative day the patient expired, apparently in a state of respiratory acidosis.

The findings at necropsy revealed the following. The body was that of a slightly built young adult female. The heart weighed 300 gm. There

FIG. 4. Close-up view of subendocardial hemorrhage and pulmonic valve.

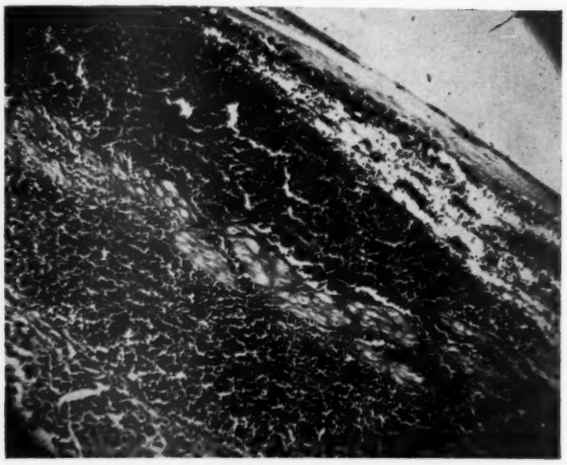

FIG. 5. Superior aspect of the interventricular septum showing accumulation of blood beneath the endocardium. Note entrapped degenerated myocardial fibers; hematoxylin and eosin, × 85.

was a 3 cm. incision, closed by sutures, over the mid-anterior portion of the pericardium. The underlying epicardium was adherent to this area by thin, easily separated adhesions. There was a 6 mm. vertical scar, closed by two sutures, on the anterior right ventricle situated 6 mm. from the interventricular septum and 2.5 cm. below the auriculoventricular groove. There was a striking hypertrophy of both chambers of the right side of the heart, the right ventricle measuring 15 mm. in thickness, and the right auricle 4 mm. (Fig. 2.) There was marked hypertrophy of the papillary muscles of the right ventricle with corresponding thickening and shortening of the chordae tendineae. The right auricle was strikingly dilated. The myocardium of all chambers was firm and had the usual beefy reddish brown color. The left ventricle measured 10 mm. and the left auricle 1 mm. in thickness. Underlying and extending just superior to the mid-portion of the posterior leaflet of the tricuspid valve, there was a 1 by 0.5 cm. roughened and granular area which contained small petechiae. (Fig. 2.) It was situated directly opposite the point of operative incision in the right ventricle. On the left ventricular side of the interventricular septum, directly opposite this area, there was a 1.3 cm. area of subendocardial hemorrhage. (Figs. 3 and 4.) This area was situated just beneath the left posterior cusp of the aortic valve and communicated with that on the right side at the base of the valve. The aortic valve measured only 4.5 cm. in circumference. The three semilunar valves of the pulmonic orifice were fused together to form a dome with a 2 mm. central perforation. A lateral slit had been made surgically, almost to the edges of the valve attachments. (Figs. 3 and 4.) The circumference of the pulmonary valve was 3 cm. The foramen ovale had a valve-like flap opening in its superior aspect. The underlying opening measured 1 cm. in diameter. There was a striking dilatation of the left pulmonary artery beginning 3 cm. from its origin and extending to the lung root. It measured as much as 6 cm. in diameter. Other significant gross pathologic findings were an intensely hemorrhagic appearance of the left lung, a red and yellow mottled appearance of the liver similar to a nutmeg, several follicular cysts in each ovary and moderate brain swelling.

Histologic sections of the interventricular septum in the region of the trauma showed large zones of accumulation of blood beneath the endocardium, separating it from the myocardium. (Fig. 5.) In some areas entrapped myocardial fibers showed marked degenerative changes, with swelling and vacuolization and disintegration of the fibers. (Fig. 5.) In other areas there were perivascular accumulations of red blood cells in the underlying myocardium which communicated with the areas of subendocardial hemorrhage. Scattered early organization of blood was also noted. Myocardial fibers subjacent to the hemorrhage showed degenerative changes due to pressure, with vacuolization and swelling of many of them. Numerous sections through this area of hemorrhage failed to show any nerve tissue. Sections of the right auricle and ventricle showed marked hypertrophy of myocardial fibers. The

pulmonic valve was composed of dense fibrous tissue showing scattered areas of hyalinization. Remaining sections of the heart were not remarkable.

Other histopathologic changes included marked acute and chronic passive congestion of the lungs and liver, several follicular cysts of various sizes in each ovary, and considerable infiltration of lymphocytes in the mucosa of the vagina. There was considerable cerebral edema, as evidenced by marked enlargement of the Virchow-Robin spaces and perineural fasciculation. In addition, scattered neuronal cells in the cerebral cortex of the frontal, temporal and occipital lobes showed degenerative changes and areas of satellitosis.

COMMENT

From the findings at necropsy it is possible to postulate the events which occurred in the cardiovascular-pulmonary system which produced the pre- and postoperative signs and symptoms.

The preoperative cyanosis of fingers and toes was due to the shunting of a small amount of blood through the patent foramen ovale. With exercise this amount of shunted blood was increased as a result of the increased right ventricular pressure.

During operation the trauma caused by the dilator impinging on the ventricular wall interfered with the A-V conduction system in the region of the superior aspect of the interventricular septum, resulting in temporary cardiac arrest which was followed by heart block. Although nerve tissue was not demonstrated histologically in this vicinity, it is assumed that the bundle of His was directly traumatized or compressed. Hemorrhage occurring in this region produced myocardial degeneration and readily accounts for the failure of the patient's heart to be restored to normal rhythm following operation. The postoperative cyanosis was a result of the heart block causing increased intra-atrial pressure with increased shunting from right to left.

It is doubtful that the hypoplasia of the aorta had any effect on the clinical course.

The cerebral edema and mild toxic encephalopathy are readily explained on the basis of terminal shock and hypoxia.

SUMMARY

The clinical and pathologic features of a seventeen year old white girl with congenital stenosis of the pulmonary valve, who died three days following valvulotomy as the result of complete heart block, are presented. This was due to accidental traumatization of the superior aspect of the interventricular septum in the region of the bundle of His with resultant localized subendocardial hemorrhage and myocardial degeneration.

Acute Appendicitis with Abscess Formation As Complication of Carcinoma of the Cecum

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ACUTE appendicitis as a complication of carcinoma of the cecum is unusual and as a subject entity has received scant mention in the literature. Robinson and Ernst[10] in 1953 were able to collect only nineteen recorded cases and added one additional case report. In the case reported herein the patient was operated upon for what was believed to be appendicitis with abscess formation. She was found to have a pericecal abscess, the contents of which contained flaky material in which adenocarcinoma was found microscopically. Subsequent right hemicolectomy revealed a small ulcerating carcinoma of the cecum which was believed to have instigated obstructive appendicitis with rupture and abscess formation.

CASE REPORT

E. M. first entered the hospital on January 3, 1953, presenting herself as a fifty year old white married gravida 11, para 11. Her chief complaint was pain in the right lower abdomen. About ten days prior to this hospital admission she began to experience sharp and aching pain in the right lower abdominal quadrant. The pain had remained localized without radiation. Throughout the present illness she had continued to have this constant aching pain in the right lower quadrant and had experienced some nausea and vomiting. There had been associated anorexia and some fever. Her last menstrual period was a normal one and had occurred two months prior to the onset of the present illness. She had had no bleeding since that time. The past history, family and social history were irrelevant.

Physical examination revealed a blood pressure of 135/80, temperature 99.8°F., pulse 90 and respirations 20. There was moderate injection of the nasopharynx. Examination of the abdomen revealed a tender, ill-defined mass in the right lower quadrant. The pelvic examination confirmed this right lower quadrant mass. Other physical findings were not abnormal. Laboratory examinations revealed the urine to be normal. Hematology revealed an anemia consisting of 8.0 gm. per cent hemoglobin with 3.60 million red blood cells; the white blood count was 14,500 with a shift to the left. Blood serology was negative. X-ray examination of the colon after barium enema revealed the following: "The colon fills readily and there are no defects seen. During the fluoroscopic examination there was a pressure deformity of the cecum, but when the cecum is completely filled, this deformity disappears."

The diagnosis was somewhat in doubt after this evaluation, it being thought that the patient had either an appendiceal abscess or a right ovarian cyst. Due to the upper respiratory tract infection and the paucity of toxic symptoms it was thought advisable to postpone surgery until a slightly later date.

The patient was treated rather intensively with antibiotics as an outpatient and was readmitted to the hospital three weeks later. Examination of the abdomen revealed considerable voluntary and involuntary muscle rigidity in the right lower quadrant and there was a suggestion of an ill-defined mass in the right lower quadrant. There was slight rebound tenderness. Pelvic and rectal examinations were within normal limits and the remainder of the physical examination was not remarkable. Hemogram on this admission was 8.0 gm. per cent hemoglobin with 3.51 million red blood cells. The white blood count was 11,050 with an essentially normal differential count. On examination the urine was normal.

Preoperative blood transfusions were given, and two days later the abdomen was explored through a McBurney incision under general anesthesia. The previously noted mass was found to be retroperitoneal and retrocecal, and

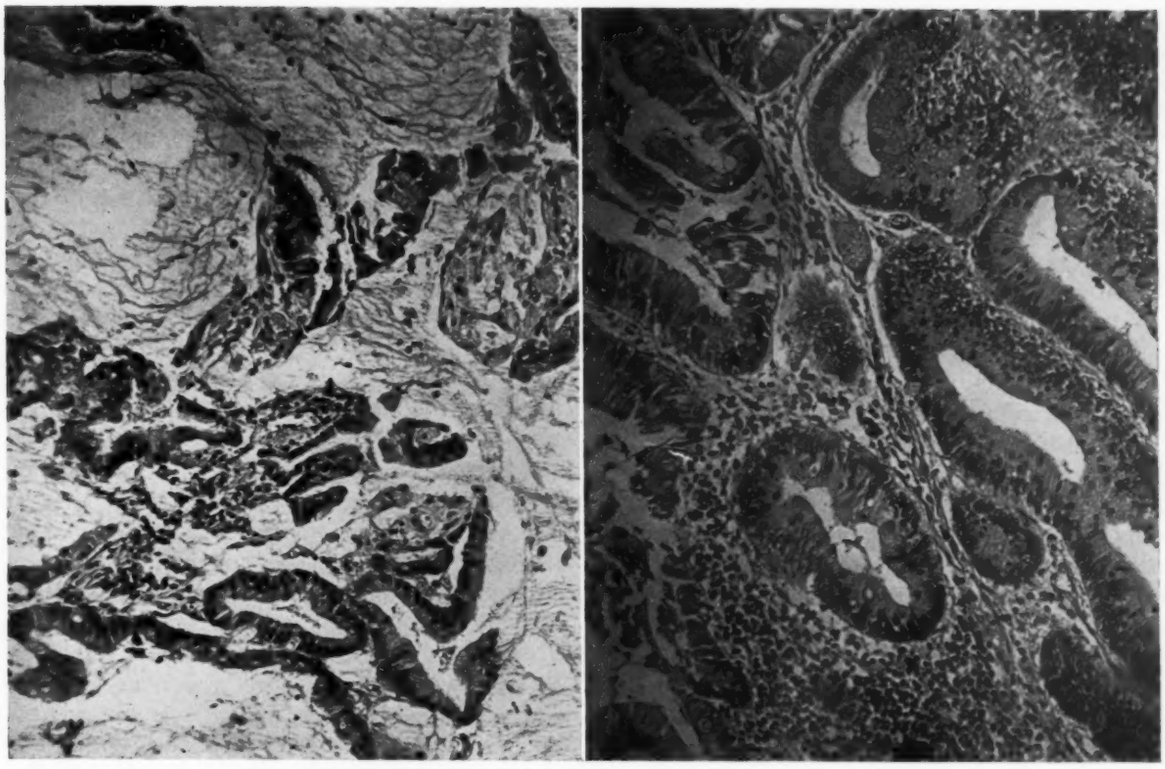

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FIG. 1. Illustrates the microscopic appearance of the mucinous flakes present within the abscess cavity.

FIG. 2. Illustrates the microscopic appearance at the region of ulceration of the cecum.

with mobilization was found to be a pericecal abscess. The suppurative content, however, was found to contain an admixture of mucoid material, and several large flakes of this along with a portion of the abscess wall were sent to the laboratory for microscopic study. The abscess cavity was evacuated and a rubber tissue drain inserted for external drainage. No evidence of an appendix was found at this operation.

The pathologic report read as follows: "The specimen consists of varied-sized masses of mucous and purulent exudate and a single piece of fat removed from the region of the cecum. The tissue appears ragged and hemorrhagic. Microscopic pathology shows a marked acute and chronic inflammatory reaction and one margin appears to have been the wall of an abscess. In this tissue, there is no evidence of any malignancy. Histologic examination of the mucoid material found in the region of the cecum reveals the presence of a mucinous carcinoma. Nowhere in this section is there evidence of any structure resembling appendix. A simple mucocele of the appendix would not have the apparent neoplastic cells in the mucoid material. With the finding at operation of normal ovaries, it is supposed that this represents a mucinous carcinoma of the cecum." Figure 1, illustrates this microscopic picture.

Postoperatively the patient was treated with nasogastric suction for twenty-four hours. The tissue drain was progressively removed, and following its removal the wound promptly closed. Eight days later, under spinal anesthesia, a right hemicolectomy with iliotransverse colostomy was carried out without incident. The pathologic report read as follows: "The specimen consists of a portion of the terminal ileum, the cecum and the ascending colon. The pericecal fat is markedly indurated and below the level of the ileocecal valve, there is a ragged perforation that extends into the cecum. The appendix is not found in this specimen. On opening the bowel, the portion of the ileum appears normal. Below the region of the ileocecal valve on the cecal side, there is a small, oval, $2\frac{1}{2} \times 1\frac{1}{2}$ cm. ulcerating tumor. The margins of the tumor are flattened and the base communicates with the perforation. The wall of the tumor is soft and mucoid.

The mesentery is free of lymph nodes. The mucosa of the cecum and colon are not otherwise remarkable. Microscopically, at the site of the tumor, the muscularis and serosa are thickened and fibrous. On the serosal surface there are fibrin and acute inflammatory cells. In the muscularis at the area of ulceration, there are lakes of mucin in which there are neoplastic, epithelial cells and distorted glands. There is an abscess in the pericecal fat that is free of neoplastic cells. Diagnosis—perforating, mucinous adenocarcinoma of the cecum." Figure 2 represents a microscopic section taken from the region of the perforation.

The patient did well postoperatively and was discharged on the sixteenth postoperative day. She was admitted for the third time eight months later with a complaint of a progressive increase in the size of the abdomen and an irregular, vague type of lower abdominal discomfort of three weeks' duration. Under spinal anesthesia an exploratory laparotomy was carried out, revealing large bilateral Krukenberg tumors and carcinomatous implants throughout the peritoneal cavity. A bilateral salpingo-oophorectomy and a greater omentectomy were performed. The pathologic report read as follows: "The specimen consists of a large amount of mucoid material that weighs 2,650 grams. Incorporated within the mass are both ovaries and both oviducts. Attached to the surfaces of the ovaries and oviducts and in some areas invading the stroma, there is a mucinous carcinoma. The tumor is made up of large lakes of mucin, in which there are scattered clumps and strands of only moderately pleomorphic neoplastic cells. The tumor is similar to that seen at the time of resection of the colon. Diagnosis—secondary mucinous carcinoma of the peritoneum and pelvis." The patient's postoperative course was essentially uneventful and she was asymptomatic when seen two months after her last operation.

COMMENTS

Because of the early non-symptomatic growth of many carcinomas of the cecum, acute appendicitis or an abscess in the right lower abdominal quadrant may be the first indication of this neoplasm. That obstruction of the appendiceal lumen may be the etiologic factor in acute appendicitis is a well recognized fact.[6] Collins[3] found such obstruction as a causative factor in 50 per cent of a series of 3,400 cases of acute appendicitis. Considering simply the mechanics involved of neoplastic obstruction, one would not expect the association of carcinoma of the cecum and appendicitis to be so unusual. Another causal relationship of the two conditions may be that the carcinoma becomes ulcerated and secondarily infected, subsequently contaminating the adjacent appendix. Uihlein and McDonald[11] have discussed the problem of primary carcinoma of the appendix and have pointed out that the cystic and colonic types, which are more frequently located near the appendiceal base, may first become manifest by the development of acute obstructive appendicitis.

The absence of an appendix at either operation and the failure of the pathologist to demonstrate one as such led us to believe that in our case the neoplastic invasion of the cecal carcinoma had blocked the base of the appendix. Subsequent rupture of a retrocecal appendix then led to the development of a retrocecal abscess within which the appendix' remains were completely necrotized. It is not always apparent from the case histories reported in the literature whether appendicitis had caused an abscess or whether the abscess had been caused by perforation of the carcinoma with subsequent infection of the adjacent appendix. In several cases, as in our own, the appendix has not been found in the operative specimen, it having apparently been completely necrotized in the abscess.[4,5,7,9]

In the usual case reported in the literature the patient has been operated upon for suspected acute appendicitis and the surgery ultimately performed in two stages.[1,2,4,8] Two factors mitigate against the recognition of the presence of carcinoma of the cecum as an underlying process in acute appendicitis: (1) An underlying neoplasm may be mistaken for the varying degree of induration of the wall of the cecum that may be present in acute appendicitis. (2) It is not considered good surgical judgment to explore adjacent organs in the presence of an acute inflammatory process. Consequently, definitive surgery for the cecal neoplasm is usually performed at a subsequent operation following microscopic diagnosis of the neoplasm.

SUMMARY

Acute appendicitis as a complication of carcinoma of the cecum is unusual. Etiologic

factors and some of the problems of recognition and therapy involved are briefly discussed. A case is presented in which an appendicular abscess was drained, the associated neoplastic process in the cecum becoming apparent only after microscopic study of the abscess contents.

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Permanent Survival of Homograft

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Homografting is as old as the concept of skin grafting. In the early part of the present century when the use of free skin grafts became popular the difference between homogenous and autogenous grafts was not appreciated. Grafts of all types were used and successful homotransplants were reported frequently. However, the evidence presented in these claims is not convincing and the only proven homograft takes have been in identical twins.[1] The following case report is on a homograft which at this writing has survived eleven months and which shows no indication of sloughing.

1 2

Fig. 1. Photograph taken on thirteenth postoperative day. All grafted skin on left extremity is homograft.

Fig. 2. Photograph taken five months after homotransplantation.

CASE REPORT

H. S., a nine year old Negro boy, was admitted to the Aiken County Hospital on December 26, 1952, with third degree burns covering approximately 32 per cent of the total body surface. The burned areas covered most of both thighs and legs. On January 10, 1953, dèbridement of the burned areas was done in the operating room under general anesthesia. On January 22nd a split-thickness skin graft was done. Four strips of skin taken from the father's thighs, measuring 3 by 9 inches, were transplanted to the recipient's left thigh and leg. These grafts measured approximately .011-inch in thickness and were taken with a Brown electric dermatome. At the same time several smaller split-thickness grafts were taken from the patient's buttock and transplanted to the right thigh. Subsequent grafts, using only the patient's skin, were done on February 6, March 31, April 22, and June 25, 1953. At the time of the last graft a sliver of skin, which incorporated both the homograft and the patient's normal skin, was taken for histologic studies. (Figs. 1 to 4.)

FIG. 3. Photomicrograph of section of normal skin.

FIG. 4. Photomicrograph of section of homograft.

During the course of treatment the patient received three transfusions of the father's blood as well as blood from the blood bank which was given during surgery.

The medical treatment consisted mainly of the following: (1) antibiotics; (2) blood; (3) ascorbic acid; (4) testosterone, 10 mg. daily for ten days following first graft; (5) kutapressin,® ½ cc. daily for twenty days following first graft; (6) multiple vitamins daily; (7) protein supplement daily; (8) elixir of benadryl,® 1 dr. three times a day beginning on February 6th and continuing until dismissal from hospital on July 16th; (9) demerol® for acute pain; (10) tetanus antitoxin; (11) furacin® dressings to grafted areas; (12) aluminum foil dressings to donor sites.*

The histologic report was as follows: "Section of skin some 4 by 0.4 cm. One segment of essentially normal structure except for an occasional focus of slight perivascular chronic inflammatory cell infiltrate (recipient). In the remainder, the epithelium is intact with some areas of atrophy of moderate degree. There is uniform ironing out of the rete pegs, producing a flat basal zone. These areas are associated with some fibrous reaction in the superficial corium and perivascular and periadnexal chronic inflammatory infiltrate. There is degeneration of adnexal structures; foreign body giant cells are oriented about occasional hair shafts in which there has been degeneration and disappearance of the follicular epithelium."

On laboratory examination the urine was negative. Hemoglobin was 81.1 per cent. Both the patient and the donor of skin had blood type O, Rh positive. White blood cells and differential were normal.

* Aluminum foil furnished by the Reynolds Metals Company, Louisville, Ky.

COMMENTS

Between the eleventh and twelfth weeks the homograft began to undergo gross changes. The graft lost some of its pigmentation and began to have a glassy appearance. There was a little sloughing around the periphery where the donor skin did not touch the patient's unburned skin. After the bandages were removed and the patient became ambulatory, much of the pigmentation returned. The left thigh which received the donor skin became

slightly edematous several weeks postoperatively. Much of this edema subsided when elixir of benadryl was started.

In reviewing the literature on homotransplantation of skin it was discovered that Caby[2] of Corbeil, France, reported a successful permanent take of a homograft from mother to daughter.

In conclusion, successful homografts are possible under some conditions.

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According to R. H. Smithwick, excision of the distal half of the stomach combined with complete vagotomy in the surgical treatment of duodenal ulcer gives better clinical results than radical subtotal gastrectomy plus vagotomy. In the latter series only 64 per cent exhibited persistent achlorhydria, whereas if a larger portion of the proximal stomach was left and it was combined with a thorough vagotomy, persistent postoperative achlorhydria, after all tests, was present in 93 per cent of the patients. In fact, even simple gastroenterostomy combined with vagotomy gave superior clinical results to radical subtotal gastrectomy with or without vagotomy. (*Richard A. Leonardo, M.D.*)

Typhoid Fever As a Postoperative Complication

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THE low incidence of typhoid fever, especially in heavily populated American communities, has practically made this disease a medical curiosity. Today's medical student may never see one case of typhoid in his four years of training, yet the existence of the disease should not be minimized as demonstrated in the following case report. This case is unique in that typhoid fever occurred as a complication following surgery.

CASE REPORT

W. F., a 44 year old white man, was admitted to Lankenau Hospital on September 22, 1953, complaining of "discomfort and pressure in the right groin." This was especially prominent upon heavy lifting. The symptoms had been present for six months, and shortly before admission a company physician had advised the patient to have a repair of a right inguinal hernia.

The patient's medical history included three hospitalizations at other institutions. The first admission, in 1929, was for a left inguinal hernia which was repaired followed by atrophy of the left testicle. In 1941 the patient suffered loss of appetite, vague abdominal discomfort, vomiting and melena. Hospital studies at that time revealed a bleeding duodenal ulcer, and a medical regimen was instituted which was temporarily successful. Acute perforation of the duodenal ulcer occurred in 1949, which required emergency surgery and oversewing of the perforation. From this period until the time of the present admission, however, the patient suffered with recurrent episodes of epigastric pain which were occasionally incapacitating and refractory to strict ulcer regimen.

The patient revealed no history of tuberculosis, diabetes, gonorrhea, syphilis, typhus or typhoid fever. He had been born in Germany where he lived until 1933 when he became an American citizen.

Systemic review was non-contributory except for the gastrointestinal symptoms noted heretofore, and these had been minimal within recent weeks.

The patient's wife and two children were living and well. No history of familial disease was elicited. The patient worked as a pattern maker in a steel plant, smoked moderately, did not indulge in alcohol and lived in a small community some distance from the hospital.

Physical examination revealed a well developed, slender white man appearing to be in his forties and in no apparent distress. Blood pressure was 118/78, pulse 72, respirations 20 and temperature 98.3°F.

Physical findings of note included a healed right upper abdominal scar and a healed left inguinal scar. There was no tenderness to palpation throughout all abdominal quadrants and no masses were palpable. The right inguinal region presented a small indirect hernia, easily demonstrable on coughing. The left groin presented no recurrence of a hernia, and the left testicle was atrophied. Rectal examination revealed slight symmetric enlargement of the prostate gland.

The laboratory studies demonstrated 5,310,000 red blood cells per cu. mm. with 15.5 gm. of hemoglobin; white blood cells totaled 8,600 per cu. mm. of which 72 per cent were neutrophils, 26 per cent lymphocytes and 2 per cent eosinophils. The urinalysis was not remarkable and serology was non-reactive for Kolmer-Wassermann tests.

The day following admission the patient was operated upon and a right indirect inguinal hernia was repaired without incident. His temperature rose to 100.4°F. on one occasion the day following surgery, after which time it returned to normal and remained so for the next six days. During this period of con-

valescence, however, an acute exacerbation of peptic ulcer symptoms developed, characterized mainly by gnawing epigastric pain. The patient was slightly relieved by antacids and a bland ulcer diet with frequent feedings. Owing to the persistence of the pain a barium study of the stomach and duodenum was made five days following operation, which revealed pronounced gastritis and a very large ulcer crater deforming the first portion of the duodenum. In view of the long ulcer history and previous complications of bleeding, perforation and recurrent pain, surgery was advised.

Accordingly, seven days following the hernia repair subtotal gastrectomy was performed for a large duodenal ulcer, removing four-fifths of the stomach and the ulcer in the duodenum. A Polya type antecolic anastomosis was performed. Preliminary abdominal exploration revealed no gross pathologic disease of the gallbladder or other viscera except the duodenum. The operation was performed without technical difficulties. A pint of blood was slowly administered during the procedure, and the patient left the operating room in good condition.

Following this operation, the patient convalesced uneventfully for seven days, his highest temperature reaching 100.4°F. on the second day after surgery. The temperature gradually declined to normal by the fifth postoperative day and the clinical course was excellent until the eighth day. By this time the patient was ambulatory, quite comfortable, enjoying a light diet and was making preparations for his discharge from the hospital. On the evening of the eighth day, however, his temperature rose to 100°F. It proceeded to rise during the next forty-eight hours to a peak of 104.4°F., after which it followed a spiking pattern for the ensuing sixteen days. Tachycardia accompanied the initial rise in fever only to change in forty-eight hours to a relative bradycardia. (Fig. 1.)

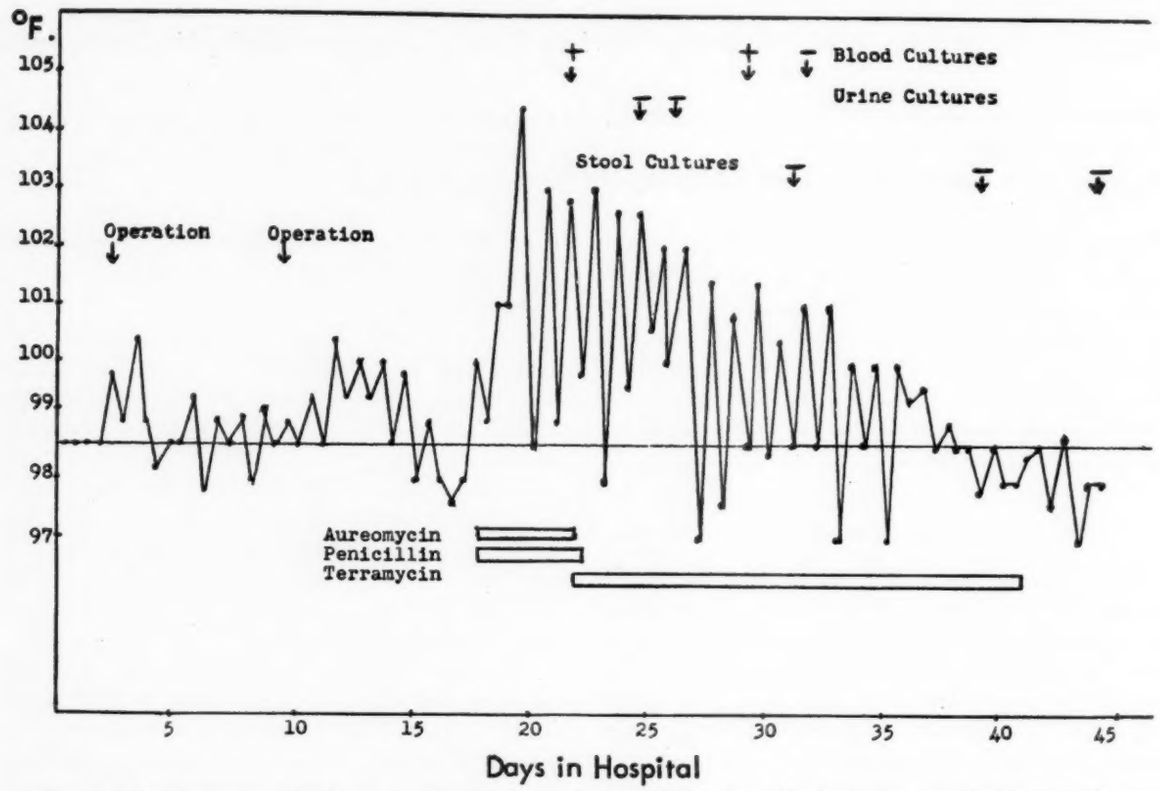

FIG. 1. Chart representing daily temperature variations, periods of antibiotic therapy and culture results.

Subjectively, the patient complained only of malaise, headache, photophobia and "feeling hot." He presented no abdominal complaints throughout the remaining course of his disease, and he was able to maintain his nourishment entirely by mouth. The most logical causes of postoperative morbidity were repeatedly investigated but there was no evidence of wound

infection, subdiaphragmatic collection, phlebitis, urinary tract infection or chest involvement.

X-rays of the lung fields revealed bilateral emphysematous lungs, but otherwise no abnormalities were detected. Under fluoroscopy the diaphragm moved freely on both sides and through an entire normal range of respiration. Careful inspection of the skin at no time revealed any evidence of a rash of any type, and the buccal mucosa was free of suspicious lesions. No lymphadenopathy was palpable in any region of his body.

Laboratory studies were of special interest and provided the means for definite diagnosis. Blood agglutination tests on October 14, 1953, were carried out revealing Typhoid O agglutination at 1 to 640 dilution and Typhoid H at 1 to 80 dilution. Five days later the O antigen agglutinated at 1 to 960 dilution and the H antigen at 1 to 160 dilution. No other agglutinations were seen excepting for Brucella abortus in a dilution of 1 to 80 on two occasions. The last agglutination test revealed no agglutination reaction for this organism.

Serial blood cultures were taken and found to be positive for Salmonella typhosa on October 14th and 16th. Sensitivity tests were carried out against the various antibiotics. This particular strain was sensitive to chloromycetin,® aureomycin, terramycin® and furadantin.® The organisms were resistant to penicillin, streptomycin and erythromycin. Early in the course of the disease the patient had been placed empirically on aureomycin when the diagnosis was undetermined, and 23 gm. of this drug were administered to him over a period of five days. In the later stages of the disease therapy was shifted to terramycin, of which he received a total of 34 gm. This change was made owing to the fact that the temperature had not declined as rapidly as we desired.

Repeated complete blood counts did not demonstrate any drop in the red cell count, nor did the white cell count rise above 9,200 cells per cu. min. The differential remained within normal range. Two sedimentation rates were quiescent during the height of the fever.

Clinically, the acute phase of the disease covered a period of approximately twenty days, and the patient was discharged after having had negative stools for three consecutive weeks. Blood and urine cultures were also free of organisms before his release. Outside of marked weakness and malaise, the patient presented no complications from typhoid. This was undoubtedly due to the liberal use of antibiotics.

COMMENTS

The origin of this isolated case of typhoid fever is still undetermined. Local health authorities were notified when the diagnosis was made, and the patient was kept in isolation for the duration of his stay with infectious precautions being observed. All hospital personnel in contact with the patient received "booster" injections or a course of three typhoid immunization injections depending on their requirements.

Close questioning of the patient revealed a possible source of infection in the rural community in which he lived. Having no municipal sewer, outhouses and cesspools were common, and drinking water was derived mainly from well water on the premises. The wells, however, were situated at least 100 feet from the latrines, as prescribed by the local health authorities. Examination of the patient's well water revealed no pathogens. Stool cultures of hospital food handlers were also investigated but revealed no typhoid organisms. The donor whose blood was used during surgery was likewise examined, with negative results.

Assuming then that the incubation period for typhoid fever is ten to fifteen days, it should be noted that the patient was hospitalized eighteen days before the disease assumed its acute stage. Another possibility is that the patient himself was a "carrier" and the stress of surgery with inadvertent pressure against the gallbladder may have liberated bacilli into his intestinal tract. Antibiotics, meanwhile, may have sterilized the intestinal and urinary tracts so that negative stool and urine cultures were obtained. The case is unusual in that only positive blood cultures could be obtained, and the stools and urines were always free of pathogens. A skin rash, also, was never present.

The patient will be followed up closely by his family doctor and by us, both from a surgical and from the "carrier" standpoint.

SUMMARY

1. A case of typhoid fever occurring as a postoperative complication is described.

2. Possibilities as to the source of infection are discussed.

Malrotation of the Intestine with Regional Ileitis

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FEW pathologic conditions of the intestine other than appendicitis and intestinal obstruction due to atresia, stenosis, adhesions, volvulus or internal herniation have been associated with cases of malrotation of the mid-gut. An unusual case of regional ileitis occurring in a malrotated mid-gut is presented. Review of the literature for the past twenty-five years does not reveal a similar case.

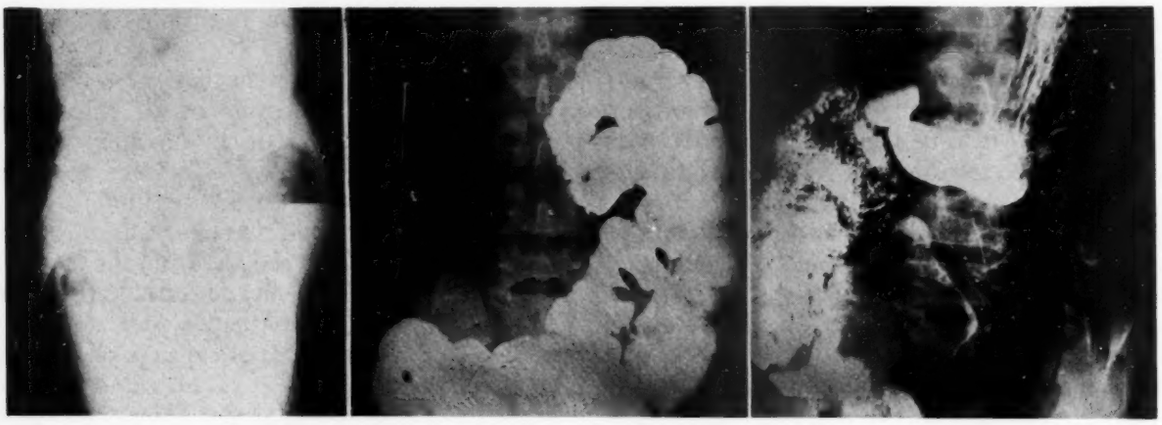

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FIG. 1. Lateral x-ray of the abdomen in the erect position shows the large anterior abdominal wall abscess.

FIG. 2. Barium enema demonstrates the colon to be entirely on the left side of the abdomen. Redundant sigmoid colon can be seen in the right lower quadrant.

FIG. 3. In the gastrointestinal series, the barium descended directly to the right lower quadrant from the second portion of the duodenum. An irregular segment of terminal ileum can be seen arching around the inflammatory mass.

CASE REPORT

A. S. (East Orange V. A. Hosp. No. 505), a twenty-eight year old white man, was admitted on December 29, 1952, with a complaint of a progressively enlarging mass of the lower abdominal wall accompanied by dull, steady low abdominal pain of three days' duration. The mass was soft, tender and non-reducible. Three months prior to admission the patient had noted a progressive weight loss of 60 pounds associated with anorexia, malaise and weakness. On a few occasions a low grade fever up to 101°F. had been present. Diarrhea had occurred occasionally in the preceding three months, but the stools were always yellow. Six years prior to admission, upon discharge from the Navy, the patient had noted two papillomatous lesions in the gluteal folds. These lesions had become painful and tender, frequently draining yellow-red malodorous material, and recently had spread throughout the intergluteal fold and perineum to the right groin.

Physical examination revealed a well developed, poorly nourished, white male in moderate distress due to the abdominal wall mass. His blood pressure was 105/75, pulse 88 and oral temperature 98.7°F. A 12 cm. tender, warm, red, non-reducible mass was palpated in the mid-portion of the lower wall of the abdomen. It was indurated around the periphery and fluctuant and tympanitic centrally. No peristalsis was audible over the mass. A moderately tender bulge was palpable in the anterior rectal

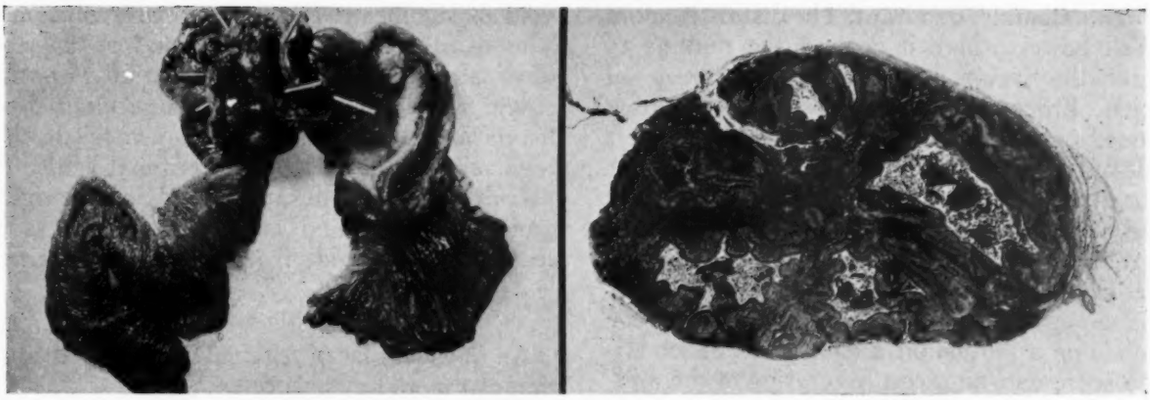

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FIG. 4. At least six fistulous tracts were traceable from the regional ileitis. Note the abrupt cessation of gross pathologic disorder at the ileocecal junction on the right.

FIG. 5. Liquefaction necrosis is prominent throughout the regional lymph nodes in the mesentery of the terminal ileum; × 6.

wall about 7 cm. from the anorectal junction. Hemogram on admission was as follows: white blood count 11,150 with 75 per cent neutrophils, 20 per cent lymphocytes, 3 per cent monocytes and 2 per cent eosinophils; red blood count 3,860,000, hemoglobin 11.8 gm.; moderate hypochromia was noted in the red blood cells. Urinalysis was negative. Total serum proteins were 8.2 gm. per cent with an albumin-globulin ratio of 0.9. X-rays of the abdomen revealed the presence of an enormous cavity with an air fluid level in the anterior abdomen above the pubis. (Fig. 1.) Incision into this cavity yielded 300 cc. of pus. Cultures revealed a mixed infection of an organism in the clostridial group, anaerobic streptococcus and a non-hemolytic streptococcus.

The skin lesions between the gluteal folds and right groin were regarded as being an infection of the apocrine glands. Skin tests with Frei antigen and purified protein derivative, stool examinations for pathogens and anoscopic and protoscopic examination up to 25 cm. from the anorectal junction were negative. A barium enema disclosed the large intestine to be entirely on the left side of the abdomen. (Fig. 2.) In a gastrointestinal series the barium proceeded straight downward from the second portion of the duodenum into the small intestine in the right lower quadrant. (Fig. 3.) Within the false pelvis it passed through a fixed, irregular segment of small intestine which was arched around the better part of a circular mass. The barium then proceeded to outline the malrotated large bowel. During the sixth postoperative week a fecal fistula developed through the healing abdominal wall defect. Installation of diodrast through a small rubber catheter inserted into the fistula demonstrated numerous thin tracts leading from the inflamed segment of small bowel. At laparotomy on February 16, 1953, the distal 40 cm. of ileum was thickened, coiled and matted together and attached to the cutaneous fistula. The mesentery of the distal small bowel was very thick and friable and contained multiple, succulent lymph nodes. Frozen section of a biopsied segment of small intestine 80 cm. from the ileocecal junction revealed normal tissue. Block resection of the distal 80 cm. of the small intestine, the enterocutaneous fistula and the proximal 15 cm. of large intestine was performed. (Figs. 4 and 5.) The pathologic report was regional ileitis with ulceration and at least six fistulous tract formations. Granulomas and liquefaction necrosis of the regional lymph nodes were present. Two plastic operative procedures were performed on the right groin, perineum and intergluteal folds for the hidradenitis suppurativa. Split-thickness skin grafts were applied with good results. One year and six months after the original hospitalization, the patient was asymptomatic and had gained 40 pounds. He was having two soft formed stools daily.

COMMENTS

In this case there was no duodenal sweep; the small intestine descended on the right side of the superior mesenteric artery to the right

lower abdominal quadrant. The distal 40 cm. of small bowel, where it crossed the midline to enter the cecum, was involved by regional ileitis. From the cecum the ascending colon passed upward on a free mesentery on the left side of the midline to a point behind the greater curvature of the stomach. Between this point and the splenic flexure it formed a short inverted loop. The descending colon and its mesocolon were fairly well fixed to the posterior abdominal wall but the sigmoid colon was very mobile on a redundant mesentery. This condition represents an arrest in rotation of the mid-gut during the tenth week of embryologic development.[1,2] No other congenital anomalies were encountered in this patient. The coincidence of regional ileitis in a malrotated mid-gut is probably fortuitous. No cause-effect relationship could be demonstrated.

Many interesting features were present in the regional ileitis itself. The past history was void of significant gastrointestinal symptoms. The disease insidiously presented as an abdominal wall abscess. The consultant in pathology[3] was impressed by the large number of fistulations and the liquefaction necrosis in the regional lymph nodes of the terminal ileitis.

Hidradenitis suppurativa involving the right groin, perineum and intergluteal fold was a separate entity aside from the regional ileitis.

CONCLUSION

An unusual case of regional ileitis in malrotation of the mid-gut is presented.

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Patent Urachus

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Patent urachus is an unusual congenital anomaly that may present a problem in both diagnosis and management. The following case illustrates several interesting aspects of this condition.

CASE REPORT

L. P., a one month old Negro male, was admitted to the John Gaston Hospital February 25, 1953, with a history of an unhealed navel which had remained moist continuously since birth. The child had been delivered at home by a midwife at term and had weighed 7 pounds. At birth the umbilicus had been considered to be unusually large. The umbilical cord had sloughed at nine days, but an unhealed reddened area had remained. The mother stated that fluid flowed from the umbilicus when the patient urinated. Three male siblings were normal.

Physical examination was essentially negative except for a moist and erythematous granulating area 2.5 cm. in diameter involving the umbilicus. A small depression 0.2 cm. in diameter was seen in the center of the granulating area. On straining or crying there was a protrusion adjacent to this area superiorly that appeared to come through the same anterior abdominal wall defect and which clearly was an umbilical hernia.

The laboratory data were essentially noncontributory except that urinalysis revealed four to ten red cells per high power field.

A catheter which was passed through the urethra in order to do a cystogram emerged from the umbilicus (Fig. 1), and the cystogram (Fig. 2) demonstrated the patent urachus. An excretory urogram showed a normal upper urinary tract.

Operation, performed March 14, 1953, revealed a fascial defect 3 cm. in diameter in the anterior abdominal wall through which a small umbilical hernia and the patent urachus protruded. (Fig. 3.) The urachus was completely patent and opened on the summit of the protrusion through the orifice previously described. (Fig. 4.)

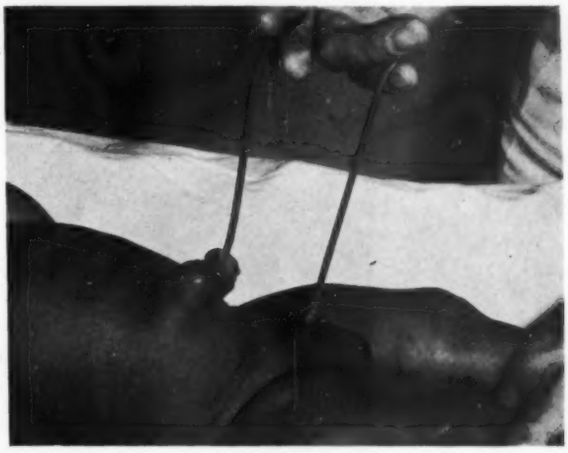

Fig. 1. Patent urachus. The catheter was introduced through the urethra and promptly emerged from the umbilicus.

A vertical elliptic skin incision was made around the umbilical protrusion, and the lower end of the incision was extended downward to the right of the midline for from 1½ to 2 cm. The rectus sheath was incised below the urachus, and an attempt was made to carry out the dissection extraperitoneally. The urachus was freed up and divided where it broadened to fuse with the bladder. (Fig. 3.) The excised urachal extension was estimated to be from 2½ to 3 cm. in length. A No. 12 Foley catheter was placed in the bladder at the point where the urachus had been amputated and was brought out through a suprapubic extraperitoneal stab wound. (Figs. 5 and 6.) A flap of the peritoneum was dissected free of the posterior surface of the bladder and the peritoneum was then closed. (Fig. 5.) The rectus sheaths were imbricated from side to side.

Postoperative complications included an episode of alkalotic tetany which was treated

FIG. 2. Patent urachus. Left: the cystogram delineates the bladder and the urachal extension to the umbilicus; right: the catheter shown in Figure 1 has been filled with radiopaque dye.

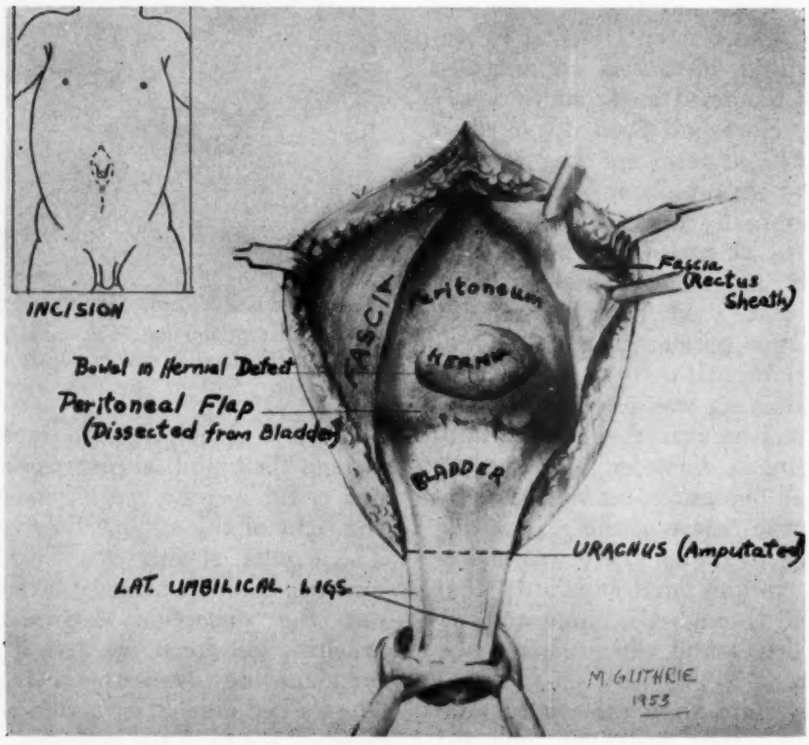

FIG. 3. Patent urachus. The artist's sketch presents the anatomic structures as visualized at operation. The umbilicus and the attached urachus have been dissected free and retracted vertically, revealing the small associated umbilical hernia. The lateral umbilical ligaments are prominent landmarks.

successfully, and a minor wound infection at the lower end of the incision. At no time did an infection of the bladder occur during or after removal of the suprapubic tube.

COMMENTS

Incidence of Patent Urachus. Although there have been over 192 cases of patent urachus reported in the literature, many of these were not completely patent throughout. Of the cases that were patent throughout, one of the most common admitting complaints was passage of urine through the umbilicus on micturition. The incidence of patent urachus is over twice as common in males as in females. Excellent descriptions of the embryology and

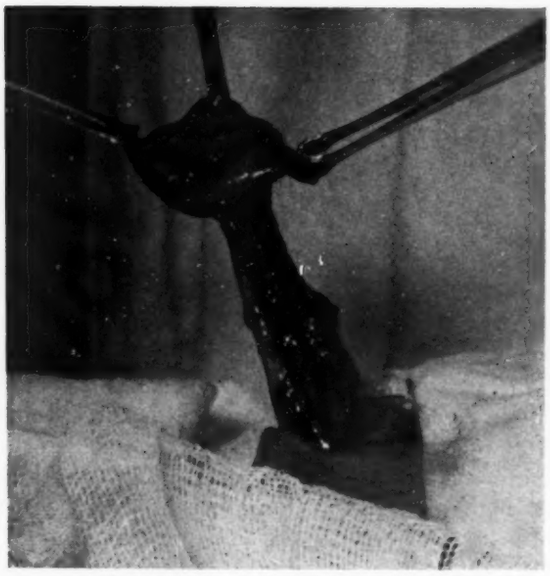

FIG. 4. Photograph taken in the operating room showing urachus with its orifice.

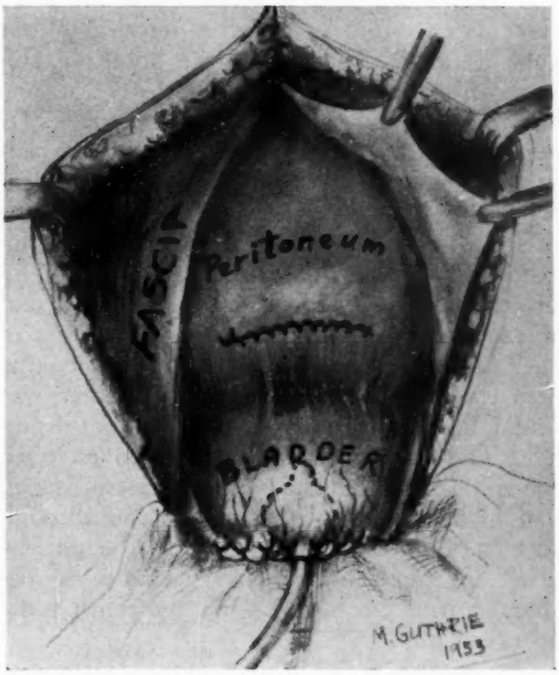

FIG. 5. Artist's sketch of the operative field following amputation of the urachus and closure of the peritoneum. The suprapubic catheter was brought out through a separate stab wound in the skin.

anatomy of the urachus have been given in the past by Cherry[2] and others.

Management of Patent Uracbus. The urachus is prone to cause infection and various types of organisms have been found and reported, including the tubercle bacillus and the fungus Actinomyces bovis. Thus surgical correction is indicated as soon as the diagnosis has been established. Cysts of the urachus have been treated successfully both by marsupialization and excision but, in view of the many adjuncts to surgery which are available today,

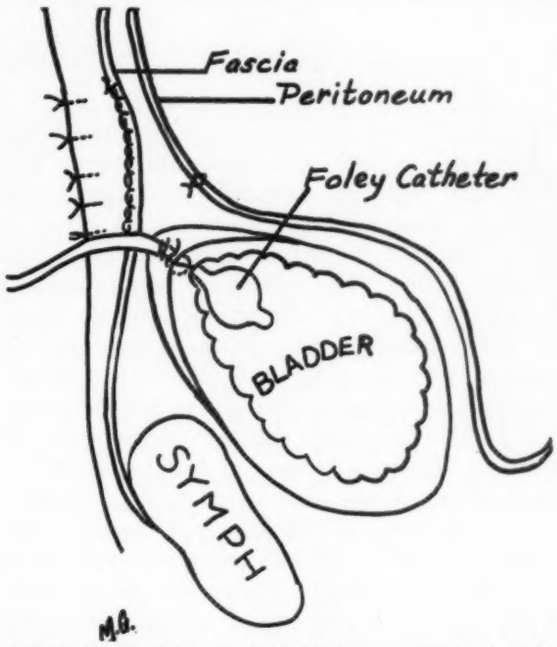

FIG. 6. Sagittal view of the bladder and anterior abdominal wall following surgery.

it appears that excision will offer the best results in patent urachus.

SUMMARY

1. A case of patent urachus in a one month old male is reported.
2. Diagnostic studies are discussed briefly. The urethral catheter inserted for cystography verified the diagnosis when it immediately presented through the umbilicus. Cystogram permitted visualization of the entire defect.
3. Surgical management is discussed.
4. Surgical excision is the treatment of choice for a patent urachus.

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Cure of Pancreatic Fistula by Pancreaticojejunostomy

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THERE is very little written about pancreatic fistula in the American literature. The first case reported, according to Garis and Merkel,[4] was by Rommelaere, a Belgian, in 1877. The first American case was reported by Pfaff in 1897. The condition was unknown before the advent of abdominal surgery. Many of the reported cases followed removal or drainage of pancreatic cysts. With the increasing number of gastrectomies being performed, the number of pancreatic fistulas is bound to increase. There are three accepted methods of treating pancreatic fistulas: drainage, excision and anastomosis to the gastrointestional tract.

There seems to be a difference of opinion as to medical treatment advised in these cases. Krois reports a case which did not improve until the patient was put on a high-carbohydrate, low-fat diet. In his case the fistula cleared up in a few weeks on this type of treatment. Wohlgemuth does not agree with Krois and recommends the diet which bears his name. It consists of very low carbohydrates but plenty of fats and proteins plus 4 gm. of soda bicarbonate one-half hour after meals. He reports on a patient on this diet in whom the fistula closed. Three other patients on this diet have been treated with success by Heineke, Koenig and Schmidt. Culler[2] reports treating two patients successfully with x-ray therapy. Hamilton reported on x-ray treatment of a pancreatic fistula which eventually closed.

However, they all agree that if the fistula does not close in a reasonable length of time, surgery should be considered.

The mortality of twenty-six reported cases of anastomosis to the gastrointestinal tract was 3.8 per cent. Reported herein is a successful case of pancreaticojejunostomy.

CASE REPORT

This patient, a well nourished middle-aged man, entered the hospital July 5, 1950. Two days before entering the hospital, the patient was stabbed in the abdomen. The wound was 2 inches to the left of the navel and 2 inches above it. The patient saw a doctor three hours after the injury, but he was not in shock so the doctor sutured the skin wound and ordered him to bed for observation. When the patient entered the hospital, the abdomen was moderately distended but soft. A Wangensteen tube was inserted for three days and the patient felt so much better that he insisted on having the tube removed. He went home the same day. Twenty-four hours after removal of the tube he again returned to the hospital. This time the abdomen was very distended and the patient was visibly in extreme shock. He complained of excruciating pain in the left upper quadrant of the abdomen. A Wangensteen tube was again inserted and three pints of blood were administered. A left rectus incision was made. The abdomen was full of bright red blood which was sucked out and the source of the bleeding, a severed artery of the mesentery, was tied off. The pancreatic area was inspected and no other bleeding was found. The abdomen was closed without drainage. The patient's condition improved until the sixth day when the wound eviscerated and had to be sutured with through and through sutures. Following this operation the patient made an uneventful recovery and was discharged from the hospital on the tenth postoperative day.

On August 6, 1950, one month after discharge, the patient again entered the hospital with marked distention of the abdomen and vomiting. A duodenal tube was inserted and twenty-four hours later a left rectus incision was made. A mass the size of a watermelon was found. The mass was opened and was found to be continuous with the lesser peritoneal sac; the wall of this sac was very edematous, fiery red and very vascular, so no attempt was made to find the source of the

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fluid, although it appeared to be coming from deep in the abdomen in the pancreatic area. A large cigarette drain was placed in the cavity and the abdomen closed. The patient made a good recovery but continued to drain pancreatic fluid. The fluid contained lipase and amylase; specific gravity of the fluid was 1,005 to 1,025, so it was evident we were dealing with a pancreatic fistula.

The patient was put on a high-carbohydrate, low-protein and low-fat diet, with 4 gm. of sodium bicarbonate administered after meals. It was difficult to measure the amount of pancreatic fluid because it seeped out around the catheter. His dressings were continually soaked. The skin was very irritated by the fluid, but tannic acid ointment seemed to correct this trouble. After being on this diet for six weeks the patient insisted on going home. He had lost twenty pounds in weight since entering the hospital.

Diodrast® was injected into the fistula tract which was found to extend to the pancreatic area. The patient returned to the office at weekly intervals. The drainage did not decrease. His weight seemed to stay about twenty to twenty-five pounds under his usual weight although his appetite was good. At the end of eight months the fistula showed no improvement under medical management so an operation was decided upon. It was hoped the fistula could be dissected out.

Under pentothal gas anesthesia the old scar was dissected out, up to the fistula tract. About 4 cm. of the tract were dissected free. So many firm adhesions and so much general oozing were encountered that it was deemed inadvisable to dissect any further. The fistula tract was large enough to admit a size 24 catheter. The distal 2 cm. of the tract was removed and an end-to-side anastomosis made, the end of the fistula being inserted into the side of the jejunum. One layer of No. 00 chromic suture was used for the first layer and interrupted cotton was used for the second. The opening in the fistula tract was large enough so that no tube was placed between the fistula and the jejunum. There appeared to be no tension on the anastomosis. A rubber tissue drain was inserted down to the anastomosis in case of leakage. The wound healed without leakage and the patient made an uneventful recovery. It is now almost three years since the last operation and this patient seems perfectly well. One objection to the anastomosis by some authors is the possibility of infection of the pancreas by way of the anastomosis; this did not happen in our case.

SUMMARY AND CONCLUSIONS

Most of the reported cases of pancreatic fistula have followed operations on the pancreas.

I believe that if a careful check were made in hospitals throughout the country, many more pancreatic fistulas would be found. Since the number of gastrectomies has increased, the injuries to the pancreatic ducts should be more frequent. Many surgeons are rather hesitant to report these accidents.

I believe these patients should be given at least six to eight months of medical treatment before surgery is resorted to, unless the patient is rapidly going downhill. In this case an early operation is indicated.

A successful case of pancreaticojejunostomy has been reported.

Pancreatic fistula transplantation is a comparatively safe operation and gives immediate improvement and a permanent cure.

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The Events Following Sudden Forced Flexion of the Knee Joint Extension-bound by Adhesion of the Quadriceps

MICHAEL BURMAN, M.D., *Diplomate, American Board of Orthopedics, New York, New York*

From The Hospital for Joint Diseases, New York, New York.

AN uncommon but logical sequence of events will be described whose common denominator was the postosteomyelitic adhesion of the quadriceps. This greatly restricted flexion of the knee, as well as the lateral movement of the proximally pulled patella. A sudden forced flexion of the joint exceeding its limited range broke the patella transversely. This was the first sequential event. The defect left after excision of the patella could be closed only by the use of a free fascial graft. A second episode of sudden forced flexion tore the repaired extensor apparatus transversely. This was the second sequential event.

CASE REPORT

In 1946, a fifty-three year old man broke the lower half of his right femur in an automobile accident. The bone became infected after open reduction and screw fixation. The foul, stinking osteomyelitis was clinically cured by extensive saucerization of the bone, but adhesion of the quadriceps limited flexion of the knee to 160 degrees.

On October 8, 1952, he made a misstep. His knee bent suddenly and so forcibly that he broke the patella transversely. The comminution of each fragment made patellectomy necessary on October 27, 1952. The expected transverse tear of the quadriceps expansions was sutured. The defect left after excision of the patella was covered by a double layer of fascia lata taken from the left thigh, since the rigid quadriceps did not allow approximation of the edges of the defect. The longitudinal wound healed well. On November 25, 1952, the joint was manipulated under general anesthesia to give flexion of 140 degrees.

The patient then left the Hospital for Joint Diseases. On November 28, 1952, he slipped in a department store. So great was the force of the sudden flexion of the joint that he tore the repaired quadriceps extensor apparatus transversely, the compounded laceration with distal convexity entering the joint. The extensor apparatus ripped again at the level of the transverse axis of the knee joint. The avulsed proximal part of the fascial graft was displaced distally.

The compounded tear was repaired the next day and the fascial graft resutured. The immobilizing plaster was removed on December 24, 1952. No grave infection followed the operation but the wound did not heal per primam despite antibiotic control. A good part of the fascial graft sloughed and was resected piece by piece. A medial subcutaneous pocket of the wound was opened on January 9, 1953, and the rest of the sloughing fascia excised. The joint was again manipulated to give flexion of 140 degrees. The wound healed slowly and the patient returned to his home in New Hampshire on February 11, 1953. He walked quite well with the aid of a cane. Weakness of the quadriceps gave him some uncertainty of power which he called "buckling," although the knee did not give way.

BOOK REVIEWS

Office Proctology. By Karl Zimmerman, M.D. Cloth binding, 92 pages, with illustrations. Pittsburgh, 1954. Reuter & Bragdon, Inc. *Price* $6.00.

This concise book was written to help the physician, who is not a proctologist, to diagnose the more common proctologic disorders and to treat those proctologic conditions that can be treated in the office with a minimum of special equipment. Only the common, easily treated conditions are included. Only one simple and time-tested method of treatment is presented for each condition. Controversial subjects are omitted. While the book is designed primarily to assist the busy general practitioner to take care of many of his proctologic patients, the medical student will also find it valuable for review.

Thoracic Surgery. By Richard H. Sweet, M.D. Illustrations by Jorge Rodriguez Arroyo, M.D. Second edition, cloth binding, 381 pages, with 159 figures. Philadelphia, 1954. W. B. Saunders Co. *Price* $10.00.

The first edition of this work was published in 1950 and was well received by the profession. This favorable reception prompted the author to finish an early complete revision ". . . which is required to provide the description of certain new technics which have gained acceptance and of alterations made in others since they were first described." Also, certain changes were made in the illustrations; three were discarded, others were altered to improve their value and some are new. The illustrations are especially good.

The author in the Preface says, "The present volume is based upon the concept that any properly qualified surgeon can acquire with relative ease a satisfactory proficiency in thoracic surgery by employing the technics herein described." Emphasis is placed on operative technic. Operations found to be useful in everyday practice are given major consideration. The author gives much space to "small details."

We think the reader would appreciate an outline of what this work embraces. We will give only the chapter headings: Surgical Anatomy of the Thorax, General Technical Considerations, Thoracic Incisions, Operations on the Thoracic Wall, Operations Concerning the Pleural Cavity, Operations on the Lung, Operations within the Mediastinum, Operations on the Esophagus, Abdominal Operations Performed through Thoracic Incisions, and Surgery of the Diaphragm.

To any surgeon interested in the subject, or who may have occasion to perform any of the present day thoracic operations, this up-to-the-minute text will not prove a disappointment.

Nash's Surgical Physiology. Second edition revised and edited by Brian Blades, M.D., with the collaboration of Edward J. Beatie, Jr., M.D., Leon Gerber, M.D., Walter H. Gerwig Jr., M.D., Ernest A. Gould, M.D., Alec Horwitz, M.D., Vincent M. Iovine, M.D., Gordon Letterman, M.D., William S. McCune, M.D., William C. Meloy, M.D., Hugo V. Rizzoli, M.D. and Jacob J. Weinstein, M.D. Cloth binding, 686 pages, with illustrations. Springfield, Ill., 1953. Charles C Thomas. *Price* $12.50.

The first edition of Dr. Nash's book was published in 1942. It had five lithoprintings, the last one in January, 1950. Dr. Nash hoped that this book would "be of help not only to the postgraduate student and the practicing surgeon, but also to the student and intern, in connection with their work in surgery." This hope has been fulfilled.

In this new edition some of the original chapters have been left intact. Others have been revised or completely replaced, bringing the work up to date. Important chapters cover the fundamentals of circulation and respiration, blood and vascular changes in shock and burns, the alimentary system, water and salt balance and the loss of gastrointestinal secretions in various pathologic states, the endocrine glands, physiology of the nervous system, acid-base balance, the liver, kidney and thyroid. These are but a fraction of the subjects covered.

Current Therapy 1954. Latest Approved Methods of Treatment for the Practicing Physician. Edited by Howard F. Conn, M.D. Cloth binding, 898 pages. Philadelphia, 1954. W. B. Saunders Co.

This is a much needed book which is indeed worth while. A thorough review would run

into the length of a monograph. It is assumed the diagnosis has been made. Knowing what ails the patient, the next step is the treatment. This large tome written by about 300 top-notch physicians gives an outline of the latest modes of treatment for diseases, ailments or pathologic conditions of the human from acne to zymosis. We do not pretend we read every word, but we did read the treatment proposed for twenty-five or thirty ailments and found them scientifically sound and up-to-the-minute. There are thirteen Consulting Editors, every one of them a national figure. Physicians of all ages, the general practitioner and specialists, will find this book valuable and satisfactory from every viewpoint.

Recurrent Dislocations. By James A. Dickson, M.D., Alfred W. Humphries, M.D. and Harry W. O'Dell, M.D. Cloth binding, 581 pages, with 78 figures. Baltimore, 1953. Williams & Wilkins Co. *Price* $4.50.

This is a well written, to the point, authoritative monograph by authors well known in their field. It will have a wide appeal to orthopedic and many general surgeons. It covers the subject in a comprehensive manner. The illustrations are good and are an aid to the reader. We read, "This compendium has been arranged to facilitate a clearer understanding of the factors involved . . . hope to stimulate a better understanding and ultimately, better treatment of this condition." The authors fulfill this blueprint. Recommended. T.S.W

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*Swartout, R., III, and Gunther, K.: "Dizziness:" Vertigo and Syncope, GP *8*:35 (Nov.) 1953.

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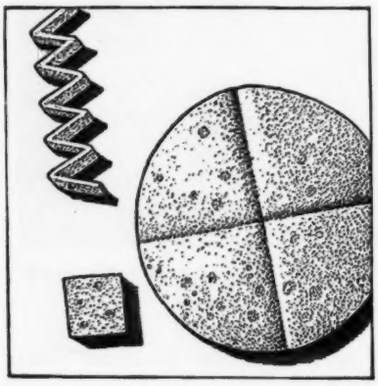

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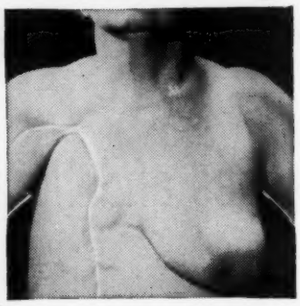

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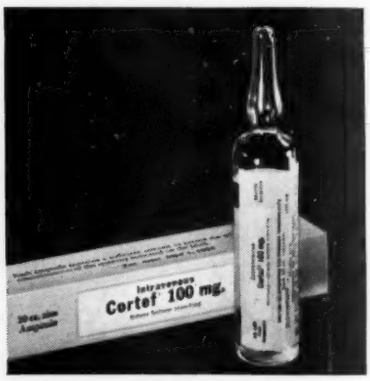

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NOTES AND QUOTES

DR. J. ROSCOE MILLER, president of Northwestern University, was elected a member of the board of directors of G. D. Searle & Co., Chicago, at the annual stockholders' meeting on April 24th. His election followed the action of the directors in increasing the number of board members from eight to nine at their quarterly meeting on January 30th.

Dr. Miller brings to his new affiliation with the pharmaceutical industry many years of active service in medicine and medical education. A graduate of Northwestern University Medical School in 1930, he became a member of its faculty in 1933 and its dean in 1941. He continued in private practice as a leading cardiologist until his election as the University's president in 1949.

He is a past president of the Chicago Medical Society and of the Association of American Medical Colleges, a trustee of the Wesley Memorial, Passavant Memorial and Evanston Hospitals, and a director of the Illinois Bell Telephone Company and the Museum of Science and Industry. His memberships include the Board of Honorary Consultants to the Surgeon General, U. S. Navy, the Committee on Veterans' Medical Problems of the National Research Council, the National Board of Medical Examiners, the Board of Governors of the Chicago Heart Association and many other academic and professional organizations.

In announcing the election of Dr. Miller to the Company's directorate, President John G. Searle also announced that net income of $1,563,282 for the first quarter of 1954 set a record high for the company. It represented an increase of 40 per cent over 1953.

It was announced by Ciba Pharmaceutical Products, Inc., Summit, New Jersey, that during the month of March $20,000 in grants was awarded for the advancement of laboratory and clinical research to investigators throughout the United States.

The following research workers and institutions were among those receiving the Ciba grants: Dr. Charles Rein, Associate Professor of Clinical Dermatology and Syphilology, New York University, Bellevue Medical Center; Dr. H. S. Kupperman, Assistant Professor, Department of Therapeutics, New York University College of Medicine; Dr. T. A. Geissman, Professor of Chemistry, University of California, Los Angeles; Dr. D. M. Green, Chief of Hypertension Clinic, University of Southern California School of Medicine; Dr. E. C. Texter, Jr., Associate in Medicine, Northwestern University School of Medicine; Dr. Morris Alex, Jewish Hospital, St. Louis, Missouri; Dr. T. L. Althausen, Professor of Medicine, University of California, San Francisco; and Dr. Louis S. Goodman, Chairman of the Department of Pharmacology, University of Utah School of Medicine.

SAVING lives is the principal function of the American Cancer Society. In pursuing its relentless campaign, the Society uses as its chief weapon a vast program of education, research and service to patients. And, to do the proper job, the Society has established a comprehensive fact-finding system, nationwide in coverage, handled through IBM methods.

"Our primary function is the planning of procedures," stated Dr. Daniel Horn, assistant director, Department of Statistical Research at the Society's national headquarters in New York. "We are available to help anyone doing research on cancer, and if machine methods are helpful we stand ready to help design a system to suit the specific purpose. We operate principally on a project basis, and our staff members are experienced in handling mass data and machine records."

With the assistance of pathologists throughout the nation cancer registration is now being taken in certain geographic areas on a statewide basis, Dr. Horn observed. Pilot surveys, such as the recent one which covered most of the hospitals in Kentucky for a study of cancer incidence, provide the basic information which may be utilized in designing other systems. Here, as in a cancer-detection study in Baltimore, mark sensing was used and the cards then were forwarded to national headquarters for processing.

One of the larger projects completed by the Society's Department of Statistical Research was the compilation of a "Manual of Tumor Nomenclature and Coding." This extensive statistical study concerned types of cancer, classifications of prognoses and other pertinent data, and is based on pathologic diagnoses

CONTINUED ON PAGE 44

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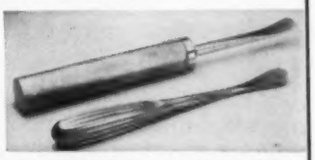

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covering approximately 70,000 cases for which cards had to be punched, sorted and coded. Now under consideration is a parallel compilation of corresponding terms for other languages to extend the study to worldwide use.

A comprehensive study now being undertaken is one to determine whether or not there is a relationship between smoking and lung cancer. Over 200,000 men between the ages of fifty and sixty-nine are being checked by about 22,000 volunteer workers in New York, Illinois, Minnesota, Michigan, New Jersey, Pennsylvania, Wisconsin, Iowa and California. In the study, controlled by IBM procedure, a carefully compiled questionnaire was used to obtain the basic information. This questionnaire was tested and revised twenty-two times before it was felt to be entirely satisfactory. Each researcher questioned about ten persons, generally relatives or friends with whom the researcher could keep in touch during the next few years for follow-up reports. The study started in the spring of 1952 and it is hoped that a definite announcement based on adequate accumulated information may be made in about a year.

The smoking study is done on a county level from which the information is cleared to the state office and from there to national headquarters. All of the original 200,000 records and most of the first two yearly follow-up reports have been processed by the Society's Department of Statistical Research. This section was organized in September, 1946, under the direction of Dr. E. Cuyler Hammond, its present director. He was joined shortly thereafter by Dr. B. Audrey Schneider and Dr. Daniel Horn, assistant directors. IBM equipment now used by the section includes two card punch machines, two sorters, a collator, interpreter, mark sensing reproducer and electric accounting machine. The broad purpose of the department is to collect, collate and disseminate information concerning the symptoms, diagnosis, treatment and prevention of cancer; to investigate the conditions under which cancer is found; and to compile statistics. Present activities may be divided roughly into four categories—statistical information, statistical consultation, field surveys and statistical research.

Another IBM procedure project is that of the Chemical Biological Coordination Center of the National Research Council in Washington, D. C. This organization is supported in part by the American Cancer Society. The Center's far-reaching punched card system lists the physical, chemical and biologic properties of many thousands of compounds. This makes possible the quick sorting of cards relating to compounds of similar properties or compounds having any desired combination of characteristics, for example, compounds which destroy mouse cancer.

Thus IBM takes its place beside the "men in white" to fight disease and save lives.

Increased survival rate of severely burned persons may be realized if results of animal research with a new drug at the University of Wisconsin can be obtained in human patients. Scientists there have shown that the drug, commonly called piperazine, will combat burn shock in animals and increase the rate of survival up to 100 per cent.

This was reported by Steven E. Jordan, Wisconsin Alumni Research Foundation research assistant in the University's department of anesthesiology. Jordan, speaking to the American Society for Pharmacology and Experimental Therapeutics, described research conducted by the departments of anesthesiology, pharmacology and pharmacy. Jordan said the survival rate for animals suffering from burn shock, but not receiving the drug, ranged from 35 to 45 per cent. One hundred per cent survival was obtained when large amounts of the drug were given following burns. The survival rate for animals receiving smaller amounts of the drug ranged from 82 to 100 per cent.

A BUDGET for 1955 of $9,500,000 has been adopted by the VII World Health Assembly, Geneva. This action represents an increase of one million dollars over this year's budget, but less than that recommended earlier by the Assembly's Committee on Program and Budget. The Committee recommendation had been for a $10,300,000 ceiling, the amount asked by Dr. M. G. Candau, director-general of the World Health Organization.

In explaining his request for an increase in the regular budget, Dr. Candau stated that the

CONTINUED ON PAGE 46

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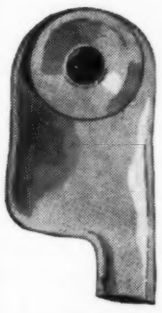

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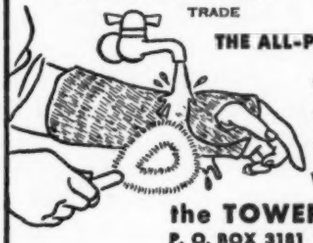

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severe cut in United Nations Technical Assistance funds allotted to WHO had obliged the Organization to postpone 176 health projects requested by governments and involving an expenditure of over $3,300,000. Since many important programs were already underway and had to be continued, he said, the decision was made to transfer those projects to the regular budget.

The real purpose of the increased budget requested, Dr. Candau continued, was to ensure the success of field activities, including joint projects with the United Nations Childrens Fund (UNICEF), to provide for normal increases and development and to cover expenses involved in increasing the use of Spanish as a working language of the Organization.

"Bearing in mind these considerations, I believe that it is fair to say that the increase asked is a modest one," said the director-general, adding that any reduction would have "a crippling effect on the orderly development of the Organization."

Earlier, the Assembly rejected a proposal made by the delegate of the U. S. A. for a smaller budget amounting to $8,607,000. In making that proposal Dr. Frederick J. Brady (U. S.) stated that his delegation was strongly opposed to any budget which would carry the U. S. contribution beyond $3,000,000, the ceiling placed by Congress on the U. S. contribution. The United States, said Dr. Brady, vitally interested in and deeply committed to many cooperative efforts to solve world health problems, did not want to find itself "forced into a position of having to depart from a long tradition of faithfully honoring such obligations."

The budget as adopted by a vote of twenty-eight to twenty-four with seven abstentions, was proposed by the United Kingdom. It is to be financed by casual income of $950,000, plus $8,550,000 assessments against Member States.

Recent studies of drug effects on mental disorders, latest progress in the chemical attack on hardening of the arteries and the role of pituitary hormones in the body are three of the topics discussed at the Fourth National Medicinal Chemistry Symposium, sponsored by the American Chemical Society's Division of Medicinal Chemistry at Syracuse University, June 17th to 19th.

Dr. Charles F. Kettering of Dayton, Ohio, dean of American automotive engineers and inventor of the first successful electric self-starter, presented the main address at a symposium banquet on Thursday evening, June 17th. Dr. Kettering has been a director and research consultant of the General Motors Corporation since he retired from active research in 1947.

Dr. Amel R. Menotti of Bristol Laboratories, Inc., Syracuse, was chairman of the symposium and Dr. Thomas P. Carney of Eli Lilly & Company, Indianapolis, was program chairman. Dr. Alfred Burger, University of Virginia, was chairman of the Division of Medicinal Chemistry.

In the past seventy-five years medical scientists have learned more about the nature, treatment and prevention of disease than in the previous 3,000 years. This point is stressed throughout "Seventy-Five Years of Medical Progress, 1878–1953," a unique symposium published by Lea and Febiger, and edited by Dr. Louis H. Bauer, F. A. C. P., secretary-general of The World Medical Association and past president of the American Medical Association. Contributors include a score of clinicians and investigators, representing every major specialty of medicine, as well as general practice. Each writer tells of three-quarters of a century of progress in his own field. As a result, light is thrown on a remarkable medical era from many angles.

The idea for the book sprang from the First Western Hemisphere Conference of The World Medical Association held last year in Richmond, Virginia. Papers contributed to this conference, presenting synopses of the history of each medical specialty, correlated with authoritative clinical summaries of advanced practice, are now gathered in this volume.

Through a grant from the A. H. Robins Co., Inc., pharmaceutical manufacturers of Richmond, presentation copies will be given to over 7,500 young doctors graduating this year from every medical school in the United States and Canada. Other presentation copies are for conference delegates from the Americas and guests of honor who represent each of the forty-eight states.

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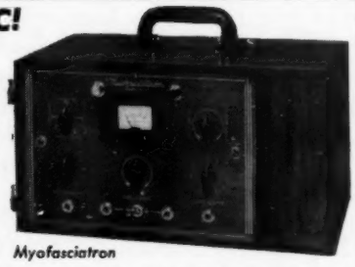

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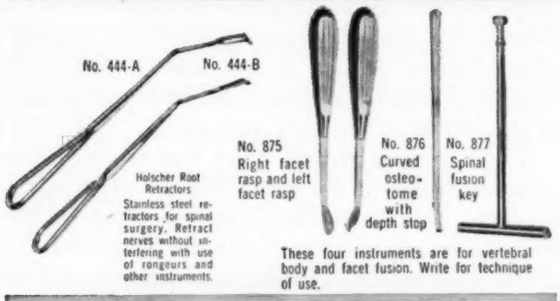
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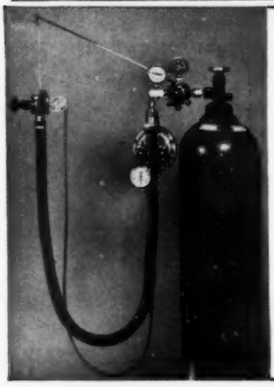

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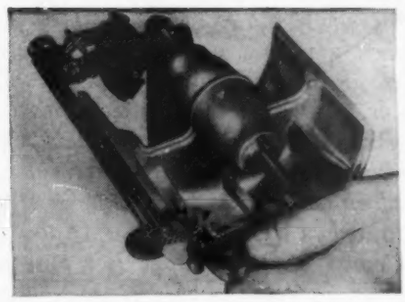

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NOTES AND QUOTES

Average length of life in the United States has reached a record high of sixty-eight and a half years, a gain of nearly four years in the past decade, according to vital statistics compiled by the Public Health Service of the Department of Health, Education and Welfare. The figures are based on death rates prevailing in 1951, and are contained in life tables published this week.

Women on the average live longer, outliving men by six years. The average lifetime expected for women at birth is 71.8 years, while the average for men is 65.9 years. This difference in the life expectancies of men and women has increased sharply since 1900, when females outlived males by an average of only two years.

White women at birth have a life expectancy of 72.6 years, compared to 66.6 years for white men. Non-white groups have a shorter average life; 59.4 years for non-white men and 63.7 years for non-white women. Although white persons outlive non-whites by an average of eight years, the difference between the two groups has narrowed since 1900 when white persons lived about fifteen years longer than non-whites.

HERMAPHRODITES, individuals with sex organs of both sexes, are also likely to have skin cells characteristic of their basic sex, according to a report to the American Association of Anatomists by Dr. Murray L. Barr of the University of Western Ontario.

Dr. Barr told the assembled anatomists about tests carried out on skin samples taken from thirty hermaphroditic patients to determine the presence or absence of chromatin. Chromatin is almost always found in the nucleus of female skin cells, seldom in male skin cells. In this way, presence or absence of chromatin gives a clue to individual sex, as carried to the embryo originally by the chromosomes or units of heredity.

Seventeen of the thirty patients tested had female-type nucleus in the skin cell. This group included three true hermaphrodites and fourteen so-called female pseudohermaphrodites. The remaining thirteen patients showed a male-type skin cell nucleus. This group included only one true hermaphrodite, one female pseudohermaphrodite and eleven male pseudohermaphrodites.

The tests, according to Dr. Barr, indicate that the rare true hermaphrodites have skin cells of either female or male type. Patients in the main group of female pseudohermaphrodites, a condition caused by overgrowth of the adrenal glands, have female-type skin cell nuclei. Male pseudohermaphrodites have male-type skin cell nuclei.

Dr. George Scatchard, professor of physical chemistry at Massachusetts Institute of Technology, whose research contributions have speeded progress in many fields of chemistry, has been awarded the 1954 Theodore William Richards Medal of the American Chemical Society's Northeastern Section, it was announced by Dr. Paul D. Bartlett of Harvard University, chairman of the section.

The Richards Medal, which was established in 1930 in honor of the late Professor Richards of Harvard, first American chemist to win the Nobel Prize, was presented to Professor Scatchard at a dinner and meeting of the Northeastern Section at Harvard on May 13th. One of the highest honors in American chemistry, the award is given biennially for outstanding achievements in research. Professor Scatchard was cited for his notable researches in the physical chemistry of solutions. He has done research on proteins, organic compounds, salt solutions and war gases, and has contributed to the understanding of how substances dissolve.

PERSONS who survive a cancer operation by several years have an encouraging life expectancy, according to a life insurance study just completed by the Society of Actuaries. Concrete evidence is produced in this study that those who have had cancer may live far into the future. This was part of a massive study of 725,000 policies covering persons with some known physical impairment, carrying through a fifteen-year period, 1935 to 1950. Included in the study were about 1,000 persons who had been operated on for cancer five years or more prior to acceptance for the insurance. Some of these persons had been accepted as standard risks, without extra premium and this group showed a mortality experience during the fifteen years just about equal to standard.

Those accepted under extra-risk policies, at an additional rate, showed a death rate about

CONTINUED ON PAGE 50

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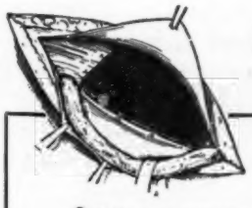

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twice the normal for standard risks, with all of the excess mortality being due to recurrences of cancer. Analysis of the experience of persons reporting a family history of cancer showed, for all sites of cancer combined, no significant tendency for the disease to be hereditary.

Tuberculosis experience was covered in another section of the study and here the actuaries report that in the case of persons with a history of pulmonary tuberculosis, underweight at time of issuance of the policy did not have an adverse effect on mortality. It had been believed previously that underweight was a distinct hazard in tuberculosis.

Among cases when the tuberculosis history was ten or more years prior to policy issuance, the death rate for selected cases with a minimum lung involvement was about normal among those accepted at standard rates and only moderately above normal for those accepted at extra-risk rates.

When the tuberculosis history was six to ten years prior to application, the death rate was normal for the standard risks and moderately excess for the extra-risk cases. Among those with a three to five year history, the death rate was twice that for standard risks. The mortality among persons with a history of chronic bronchitis was unfavorable, deaths among them being about twice the normal.

This study, the most comprehensive of its kind ever undertaken, covered 132 groups of physical impairments and medical histories, further subdivided into 388 classifications.

The over-all results, compared with a similar analysis made in 1929, shows that longevity has increased materially for persons with most impairments. The mortality among such persons has decreased, in the aggregate, in about the same proportion as that among standard risks during the past fifteen years.

Evidence that the pituitary gland is important to the growth of female sex glands was presented to the American Association of Anatomists by Dr. Marion I. Barnhart of Wayne University, Detroit. Dr. Barnhart told the anatomists how female sex glands from recently born mice speeded up in their functioning in the presence of small quantities of pituitary gonadotropin. This speed up of function was measured by checking on the amount of oxygen consumed by pooled collections of as few as five of the glands.

The pools of mouse female sex glands were placed in cultures of plasma clots, some with no hormone added, others with varying amounts of additional pituitary hormone. Oxygen consumption was then checked at ten- and fifteen-minute intervals. The glands grown on unfortified culture took up oxygen at the normal rate, or within 10 per cent of it. Those grown on cultures fortified with pituitary hormone took up from 20 to 30 per cent more oxygen in the same length of time.

Drs. William Montagna and Herman B. Chase of Brown University reported to the American Association of Anatomists interesting new evidence on the growth cycle of human hair. The two scientists from Brown traced the pattern of hair degeneration and regrowth on the human scalp after treatments with x-ray. After the x-ray treatment the entire matrix of the follicle bulb degenerates. This is followed by more gradual degeneration of the upper bulb, then by shedding of the hair. At this point the remaining follicle becomes a solid core of cells from the outer sheath of the follicle. At the same time this degeneration is taking place, the base of the follicle is retreating further into the skin. After five to seven weeks the base of the follicle is resting halfway up in the scalp skin. Now the process of regrowth begins to take place. The first sign of this is an increase of the cells at the base of the follicle cord to make contact with the skin bud adjacent to them. After growing around this bud, the follicle continues to reform and eventually grows a new hair.

The scientists point out that their observations indicate the outer sheath of the upper portion of the follicle to be most important in regenerating new hair. The lower half of the follicle is a temporary structure which must be formed anew at the onset of each hair generation.

Until recently scientists interested in the problem of hair growth generally believed that hair follicles in the "quiet" state set aside a "hair germ" to begin the process of new growth. These newest findings indicate the older theory to be incorrect.

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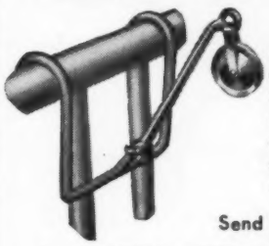

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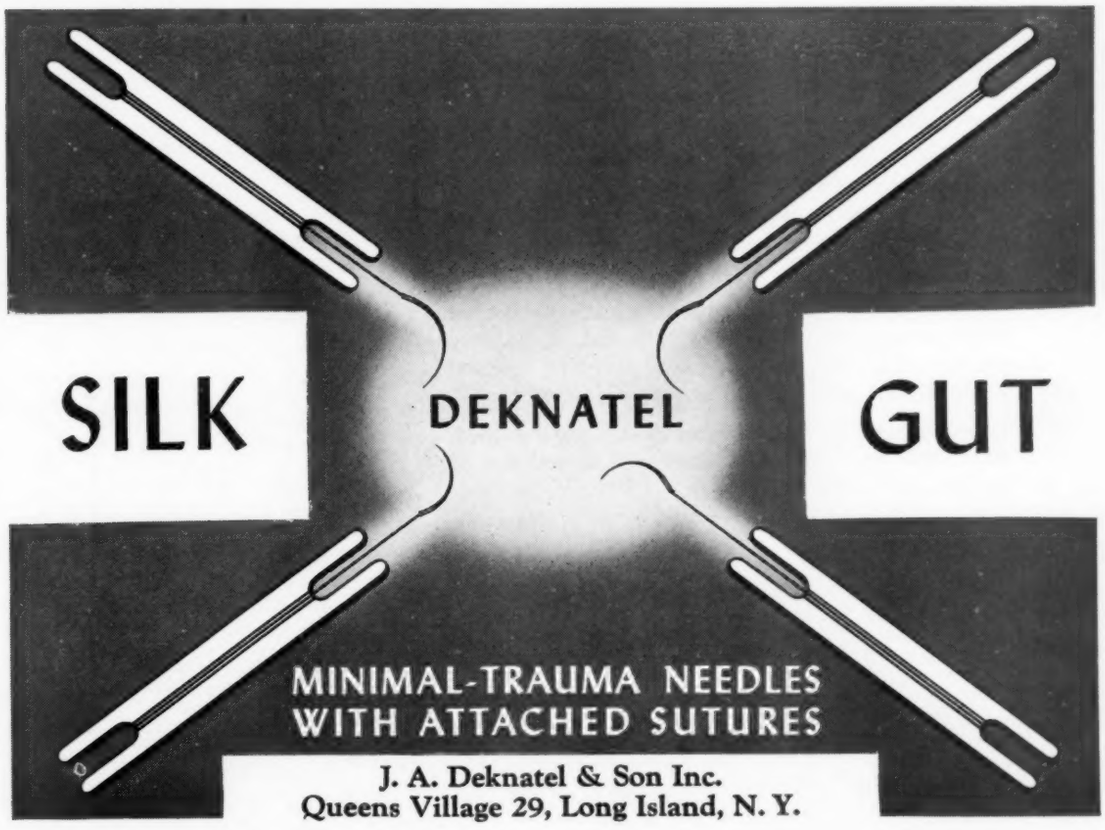

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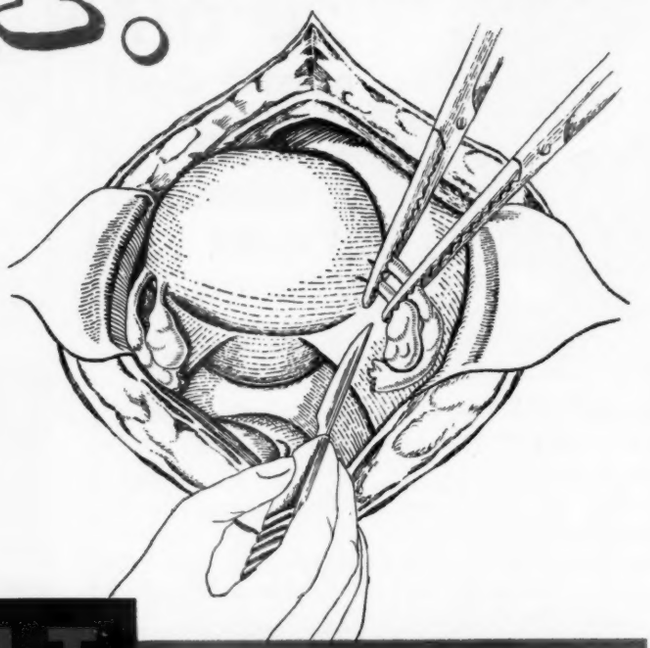

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